THE MEDICAL JOURNAL OF AUSTRALIA

VOL. I.—38TH YEAR. SYDNEY, SATURDAY, FEBRUARY 10, 1951. No. 6.

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RECENT ADVANCES IN CANCER RESEARCH.[1]

By W. E. GYE, M.D. (Edinburgh), F.R.C.P., F.R.S.,
and
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IN one of the text-books in use when we were students a neoplasm is defined as a "new formation of tissue which originates without known cause, continues to increase, comes to no physiological termination and serves no useful purpose". It is our object in this lecture to examine recent advances in knowledge of this "unknown cause", to examine what is meant by "cause" in this sense, and to point out the clinical importance of fundamental research into its nature.

The outstanding pathological feature of any neoplasm is continued cell division, either in the original tumour or in metastases derived from it or in both. The essential aim in treatment, stripped of all technical detail, is to stop this cell division. It is obvious that mechanical and physical methods represent a relatively crude attempt at this, since they are bound to destroy both normal and malignant cells and are applicable only to relatively local areas. They can never attain efficiency enough to become, not only locally selective, but generally so, that is, capable of destroying metastases whether the presence of these is known or not. The nearest approach so far made to an ideal medical treatment is the administration of œstrin in prostatic cancer, though even this does not do more than make use of a physiological property of a particular type of malignant cell. It is in no sense a cure and its administration must be continuous or the unexplained cell division will start again, and usually does in spite of it. What is required is a knowledge of the cause of the cell division, and until this is complete no direct attempt to find a truly selective "antimitotic" (if such a coinage be permitted) can be made. Substances which prevent all mitoses are obviously unsuitable, since they will be lethal from interference with bone marrow and other vital processes. One selective for malignant cells wherever they may be is required.

How much is already known of the cause of the continued mitosis? Ever since Percival Pott pointed out in 1775 the connexion between soot and epithelioma in chimney sweeps our knowledge of possible initiators of cancer has been widening until at present the list of known carcinogenic agents, ranging from heat, X rays and ultraviolet light through parasitic irritation to an ever-increasing list of chemical substances, including both exogenous materials such as the coal tar derivatives and endogenous products of hormonal nature, is by its very variety and the absence of any common factor a strong indication that these things are not the ultimate and primary cause. They can be looked on as initiators or "activators" of the disease (secondary causes), but their continued presence is not necessary for the continuance of the cell division, which must therefore have another cause, the primary or continuing cause of cancer.

That the secondary causes are not necessary to the continuance of the cell division is evidenced by the fact that an experimental sarcoma started by methyl-cholanthrene (one of the coal tar derivatives) can be transplanted from mouse to mouse for hundreds of generations, yet only from the first tumour can any methyl-cholanthrene be recovered, though, as we shall see later, something else can be recovered from the *n*th generation which will start a fresh tumour without the intervention of methyl-

[1] Read at a meeting of the Western Australian Branch of the British Medical Association on November 29, 1950.

cholanthrene. Similarly, radium will start an epithelioma, but no radio-active material is present in the metastases or in transplants.

The present position is that we are faced with a disease differing in clinical behaviour according to the organ or region attacked, yet obviously not many diseases but one, since all the clinical manifestations are unified by the microscopic pathology which shows us the essential unrestrained cell multiplication as the basis of all of them. The obvious causes are also multiple, but they appear to be inciting causes and not continuing causes, since they are recoverable only from or have acted only on the primary lesion. Looked at in the light of medical history, it is obvious that the situation is similar to that concerning tuberculosis in the period after the cellular pathologists had shown by histological study of the structure of the tubercle that all the clinical manifestations (phthisis, white swelling of the knee, cervical scrofula, meningitis *et cetera*) were one single disease, but before Koch's discovery of the tubercle bacillus explained the various riddles. It was known, for example, that tuberculosis was "caused" by knife grinding, by some forms of mining, by silica dust from stone cutting, by malnutrition, by overcrowding *et cetera*. It was also known that the disease could be passaged through innumerable guinea-pigs or rabbits, none of whom had been exposed to silica dust *et cetera*, nor from any of whose lesions could silica or anything else presumed causative be recovered. Exactly what was being passaged remained a mystery until the discovery by Koch that the tubercle bacillus was the continuing cause, ever present in all the lesions, always recoverable from them, and always handed on in transplants. The silica and other apparent causes merely acted as initiators or predisposers. We have an almost exact parallel in our present knowledge of cancer. Cancer research has passed through the phases of unification of all neoplasms as one disease on histological grounds, of recognition of animal cancer as the same as human cancer, and of investigation of an ever-growing number of initiating or "tumour-inciting" causes. The most recent advances are showing that a unifying agent, present in all the tumours so far investigated (whether spontaneous or chemically induced) and recoverable after any number of transplants, can now be separated from the cancer cell and used to start a tumour afresh without any other inciting cause, just as the tubercle bacillus alone will start the disease and keep it going. It is in this primary and continuing cause that our interest lies, since all its properties suggest that it is a virus, and since antiviral substances are just beginning to be discovered, this line obviously gives hope for the future.

Many speculations have been put forward in the past as to the nature of the continuing cause, but they all fall into two main categories: on the one hand it may be something inherent (genetic) in the cell structure which predisposes it to react by continued division to a multitude of diverse stimuli, or, on the other hand, it may be of exogenous nature, reaching the nucleus when the resistance of this is weakened by the "secondary cause" and by continuing to multiply within the cell, continually stimulating this to further mitosis, long after the secondary cause has ceased to act. That this second speculation is the correct one is becoming increasingly obvious, and as our knowledge of the properties of this extrinsic causative agent grows the possibility of ultimately achieving a direct selective chemical attack on it becomes apparent.

Four signposts pointing the way to this conclusion have long been known and their full significance is only now understood.

The first important step was made independently by Bashford and by Ehrlich. It is that a breast cancer in laboratory stock mice sometimes develops a sarcoma of the connective tissue within the mammary cancer. This observation has been repeated now many times, and since the introduction of pure line strains of experimental mice has become increasingly common. Careful histological research supports the conclusion that the malignant transformation of the stroma is due to some influence from the adjacent malignant epithelial cells. It would be difficult to maintain that no agent passes from the epithelial to the connective tissue cells, for the alternative explanation would involve us in a fantasy in which the normal cells merely copied the bad habits of their neighbours. It is much more likely, in view of recent advances in virology, that an agent adapted to existence within an epithelial cell mutates to a form which can live in a connective tissue cell (compare the neurotropic and the dermotropic forms of the herpes virus).

The second signpost on the road to the unavoidable acceptance of the presence of an intracellular agent as the primary cause of cancer is the discovery by Ehrlich in 1907 that mouse mammary cancer can be stored at a temperature of −10° C. for two years and then give rise to a tumour on injection into a mouse. The first explanation, that given by Ehrlich himself, was that the cells themselves survived and on introduction into a mouse began to divide again and so to act as a tumour graft. That this is not the true explanation is indicated by the fact that mouse mammary cancer cells are killed between 4° C. and 0° C. in a day. Why, then, should they be expected to survive at lower temperatures? It is known that mammalian embryo cells are killed by cold very quickly, yet recent work has shown that tumour cells stored at −79° C. or even lower for twelve months give rise to tumours on introduction into new mice. This matter was further investigated by Cramer in 1930, who gave cogent reasons for supposing that the true explanation was that the cold killed the tumour cells and liberated an intracellular agent which was thermolabile but capable of initiating a new tumour on introduction into a fresh mouse.

The third and greatest advance was made by Peyton Rous at the Rockefeller Institute in 1912, when he demonstrated a filterable virus as the primary cause of the cell multiplication in a sarcoma of a chicken. Since that time over two hundred virus tumours (transmissible by filtrate or dried material) in chickens, ducks and frogs have been discovered and investigated. A filterable virus papilloma of a rabbit was discovered by Shope in 1933, but apart from this it appeared impossible to transmit mammalian sarcoma or carcinoma other than by the grafting of living cells. This led to a strange dichotomy of thought. It was accepted that tumours of submammals were of virus origin, but those of mammals appeared to be in a class apart, strange, esoteric, associated with mysteries "the solving of which would reveal the secret of life itself" (Ehrlich). This led to an attitude of extreme pessimism, still rife and ending in sterile clinical observations or the search for improved surgical and radiational forms of treatment only.

The fourth signpost, the full significance of which is even now not fully realized, abolished this apparent dichotomy of tumour cause by bringing a whole class of spontaneous mammalian tumours (the Bittner adenocarcinoma of mice) into the virus category. In 1936 Bittner, at Bar Harbour, demonstrated the existence of an "agent" (since seen with the electron microscope to be a virus) which he called the milk factor, which was present in the milk of inbred strains of mice showing a high death rate from spontaneous mammary cancer. In his laboratory Bittner had built up by inbreeding over many generations two strains of mice, one of which showed a death rate among females of 95% from cancer of the breast, the other strain being practically entirely free. The obvious explanation was that a genetic factor was present in the one strain and not in the other. This was further suggested by the fact that the males of the affected stock could be made to develop mammary cancer (100%) if given œstrin for long periods, while the males of the unaffected line showed no such response to œstrin. Indeed the genetic theory of cancer had received its greatest backing from students of these inbred mice until the whole theoretical edifice of genetic constitutions and somatic mutations was destroyed by the surprising observation of Bittner that if mice of a high cancer strain were taken at birth and suckled entirely on mothers of the cancer-free line no cancer developed in them or their offspring. In other words, the apparent hereditary nature of the tumours in this line was entirely due to the passage of the milk factor from mother to offspring after birth. The reverse

experiment also held good, in that low cancer strain mice suckled on high cancer strain mothers developed cancer. The "natural history" of the Bittner virus is now known in fair detail. The virus exists in two forms, latent and active, the latent form being secreted in the milk and passing to the new-born young (compare other viruses, such as *herpes simplex*, which require an activator to produce the disease, though present in the body in latent form from early life). This latent form circulates throughout the body, being recoverable from all the organs of both male and female mice. The activator which converts it into the pathogenic form (in which it produces the endless cell multiplication) is œstrin in small doses over a number of months. The normal œstrin of the female or administration of œstrin in the male both activate the virus which then enters the mammary epithelium and stimulates it to repeated division. The active form of the virus is therefore cell specific. These facts point very definitely to a bridging of the gap between the virus tumours of chickens *et cetera* and mammalian cancer, but until recently it had not been possible to transmit a mouse cancer directly except by a living tumour graft. The latent form of the virus could be transmitted by the milk to virus-free mice, but months of waiting were necessary for the tumours to develop, since the action of œstrin over a period was necessary. The active form of the virus, that is, the form in which it is cell-specific and can induce immediate tumour formation in mammary tissue, eluded all attempts to isolate it in either filtered or dried material.

In 1948, at the Imperial Cancer Research Fund Laboratory, an intensive study of this problem, namely, the obtaining of an active agent free of living tumour cells which would be cell specific and start a new tumour (without the intervention of an activator) when injected into the appropriate region of a healthy mouse, was begun. The first line of attack was through a study of the effect of cold and other agents, lethal to normal adult or embryonic mouse cells, on minced tumour tissue. Three types of tumour were chosen, representative of all mammalian tumours (that is, sporadic sarcoma and carcinoma, and chemically induced sarcoma) and in all it was found that storage of tumour mince at −79° C. for times varying from 15 minutes to 182 days produced material which would start a new tumour (only if injected into a suitable spot) within periods varying approximately between seven and twenty-eight days. This material was therefore active and by comparison of times of appearance of tumours with times of freezing it was possible to show enhancement of activity with increase of freezing time. This is incompatible with the idea of survival of some of the cells of the frozen tumour mince, since if this occurred more cells could not be alive after twelve days than after twelve hours' freezing. It is much more suggestive of gradual death of cells with liberation of virus and perhaps also denaturation of an antibody by cold. Prolonged survival of activity in intense cold is a characteristic of almost all viruses (compare viruses of the typhus group).

We also investigated the effect of freezing and storing in various solutions and found that distilled water, strong concentrations of sodium chloride, glycerine or 40% dextrose had no effect on the activity of the frozen material, though they all prevent entirely any growth on attempted tissue culture (though fresh tumour tissue is extremely easy to grow in culture). All this indicates that freezing kills the cells and liberates a virus.

The doctrine that cancer cells multiply in virtue of some mysterious intracellular distortion originated from the work of the most famous German pathologists (for example, Virchow, Ribbert, Cohnheim) and was apparently supported by the work of such great scientists as Ehrlich and such skilful propagandists as Bashford and Ewing. This doctrine cannot be lightly disposed of and therefore it was not thought sufficient to draw the above conclusions from a study of frozen material alone, even though (as in one of our experiments) such material had been frozen at the temperature of liquid nitrogen (−190° C.) and still found to be active. We therefore realized that it was necessary to dry the tissue to dust (a procedure accepted as lethal to both normal and tumour cells) and achieve propagation with this dried material before the shibboleth of propagation of tumours by cellular division alone could be entirely disposed of.

In the past it has been found that the Rous chicken tumour and other tumours of submammals are readily propagated with dried material, but that mammalian tumours (other than the Shope rabbit papilloma) cannot be obtained in this way. Our work on the result of long freezing of tumour mince and the observation that the frozen material soon loses its activity at room temperature furnished the explanation. This new knowledge that the cancer virus (both the sarcoma and the carcinoma variant) can be handled only at temperatures below −10° C. is obviously the key to all future experiments with it and explains the failure of all the older workers (with the exception of Ehrlich and Cramer) to obtain active cell-free material. Experiments and apparatus were therefore designed to allow of the drying of stored refrigerated material at −25° C. When this was done, active dried material was obtained in 66% of sarcoma experiments and in 35% of the carcinomata. The experiments with the sarcomata yield better results for two reasons: first, the virus is not so thermolabile and will withstand rapid freezing and thawing more than once, so that if the temperature rises accidentally above freezing point during the experiment, this is not necessarily a failure; and second, since the sarcoma variant is adapted to life in any subcutaneous connective tissue cell, the inoculation can be made anywhere on the surface of the mouse and tumours appear within a month. In the case of the mammary carcinomata the virus is extremely thermolabile and will disappear if the temperature is allowed to rise above −10° C. at any time after the initial mincing and freezing. Also it can initiate a new tumour only if it is brought in contact with mammary cells, and although the breast tissue of the mouse is very diffuse there is not 100% certainty of the injection reaching any of it. When the injection is successful the tumours appear at the site of inoculation in about five weeks and in both male and female mice. This may seem strange until we remember that (unlike the milk factor) the virus is in its active form, having already been activated by the œstrin of the female mouse of origin.

These experiments on refrigerating and drying tumour cells to free an active agent having many of the properties of a virus are among the most hopeful that have ever come out of cancer research, since they point the way to the ultimate finding of a cure; that is to say, a cure in the sense that penicillin is a cure for lobar pneumonia or aureomycin for virus pneumonia—in other words, a substance having a specific action on the primary cause of the disease. Surgery, however refined, can never hope to recognize and remove every malignant cell; radium and X rays can never be made selective for malignant cells without any action on normal tissues. But now that we know that chemical substances exist with specific actions on whole groups of bacteria or whole families of viruses (for example, the typhus variants) the possibilities are great. It is lucky that this advance in our knowledge of cancer causation comes at a time when the finding of antiviral substances is a reality.

Two lines of research are open to us: one to find a substance which will kill the virus in its latent phase and so prevent the development of cancer even when the secondary causes are present, and the other to find an antibiotic which will circulate in the blood-stream and kill the active virus particles whether they are in the cells of the primary growth or in the metastases.

Summary.

1. A statement of the cancer problem in the light of the clinical pathology is made.

2. A brief historical survey of the discovery of the apparent causes of the disease is given.

3. A distinction is drawn between the primary and secondary causes.

4. The genetic *versus* the extrinsic primary cause is discussed. The work of Ehrlich, Bashford, Cramer, Rous

and Bittner as signposts on the road to the extrinsic primary cause is mentioned.

5. The question is raised of the unification of the virus tumours of birds with mammalian cancer.

6. Reference is made to recent work demonstrating the propagation of mammalian tumours with tumour material frozen to −79° C. and subsequently dried.

7. An interpretation of the results and a suggested method of attack on the problem in the light of the latest knowledge are stated.

Bibliography.

Craigie, J. (1949), "A Drying Apparatus for the Study of Tumour Transmission", *British Journal of Cancer*, Volume III, page 250.

Cramer, W. (1930), "Possibility of Transmitting Mammalian Neoplasms without Intervention of Living Cells", *Scientific Reports on the Investigations of the Imperial Cancer Research Fund*, Volume IX, page 21.

Ehrlich, P. (1907), *"Experimentelle Studien an Mäusetumoren"*, *Zeitschrift für Krebsforschung*, Volume V, page 59.

Gye, W. E. (1949), "The Propagation of Mouse Tumours by Means of Dried Tissue", *British Medical Journal*, Volume I, page 511.

———, Begg, A. M., Mann, I., and Craigie, J. (1949), "The Survival of Activity of Mouse Sarcoma Tissue after Freezing and Drying", *British Journal of Cancer*, Volume III, page 259.

Mann, I. (1949), "Effect of Low Temperatures on Bittner Virus of Mouse Carcinoma", *British Medical Journal*, Volume II, page 251.

——— (1949), "Effect of Repeated Freezing and Thawing on Mouse Carcinoma Tissue", *ibidem*, page 253.

———, and W. J. Dunn (1949), "Propagation of Mouse Carcinoma by Dried Tumour Tissue", *ibidem*, page 255.

"HEPTALGIN" AS AN OBSTETRIC ANALGESIC.

By MARIE HILL, M.B., B.S., D.A.[1]

"HEPTALGIN" is the trade-name given by the Glaxo Laboratories to their synthetic preparation of 4:4-diphenyl-6-morpholino-heptan-3-1 hydrochloride, more commonly known as C.B.11.

Pharmacologically the analgesic potency of "Heptalgin" is claimed by the makers to be six times that of morphine, twenty times that of pethidine, and at least three times that of amidone ("Physeptone"). It is stated to have virtually no depressant effect on the respiration. These claims are based on animal tests and tests on human volunteers.

Wilson and Hunter (1948) conducted a series of experiments for relative analgesic potency between C.B.11, pethidine and amidone, in which they used the method of Hewer and Keele (1948). This test method for estimating the relative analgesic potency of drugs consists in occluding the circulation of the arm of the subject by a sphygmomanometer cuff inflated to 220 millimetres of mercury, then causing the muscles to be flexed 60 times for one minute. An intravenous injection of the drug to be tested is then made into the opposite arm. Levels of pain relief are established for comparable doses of the drugs in question. By this method Wilson and Hunter found C.B.11 to be more effective than amidone and much more so than pethidine.

The toxicity of C.B.11 is also claimed to be lower than that of the other substances. This claim is borne out by Wilson and Hunter. They found the side-effects to consist of transient dizziness and a feeling of detachment. Very little cortical depression was evident. Giddiness and vomiting were noted only in the ambulant, and in no case in those confined to bed. With intramuscular administration, the relief from pain is noted in ten to fifteen minutes, the effects lasting two to three hours. With oral administration there are no hypnotic effects.

Clinical Trial.

"Heptalgin" was given by intramuscular injection to 50 patients in labour.

Forty of these patients were given "Heptalgin" alone, as the sole analgesic agent. They are discussed and then compared with four series, each of 40 similar patients. In the first of these series the patients were given intramuscular injections of pethidine 100 milligrammes, in the second intramuscular injections of pethidine 50 milligrammes *plus* hyoscine 1/300 grain, in the third orally administered doses of potassium bromide 30 grains *plus* chloral hydrate 20 grains, and in the fourth no analgesia whatever. The last series was included for the sake of comparison in regard to the condition of the infants at delivery, and to the duration of labour. All these patients were delivered under ether-air mixture from a Small's machine, given for an average of ten minutes when labour was normal and unassisted and for an average of thirty minutes for instrumental delivery. All the patients approved of the anæsthesia by this method for delivery.

The remaining 10 patients of the 50 were given "Heptalgin" in addition to various other analgesics during the course of their labour, which was of long duration in every case. They are discussed separately. In each case an endeavour was made to ascertain from the patient the relative degree of relief experienced from the different analgesics.

The 40 patients who had "Heptalgin" alone consisted of 14 *primiparæ*, 13 *secundiparæ*, five *tertiparæ*, six *quadriparæ*, one *quintipara*, and one *sextipara*. All were normal as regards their general and obstetric conditions, except three; one of these had toxæmia, one anæmia, and one anæmia *plus* pyelitis. An attempt was made to estimate their temperamental diathesis by a classification into three groups—those of calm temperament, those of average outlook with some degree of nervousness, and those who, in addition to being highly nervous and apprehensive, were uncooperative as well. Their assessments of the effects of the analgesics were classified as good, fair, and unsatisfactory.

For convenience, and comparable grouping, they have been grouped as 14 *primigravidæ*, 13 *secundiparæ* and 13 *multiparæ*.

"Heptalgin" was given at the stage of three-fingers' dilatation of the cervix, in a dosage of 10 milligrammes by intramuscular injection.

Discussion.

Of the 14 *primiparæ*, whose ages ranged from nineteen to thirty-six years, nine were delivered normally and five instrumentally. Ten of them obtained good relief from the drug, seven of them being calm, two average in response, and one very nervous. Three found the effect fair, and one unsatisfactory, the last-mentioned four being of highly nervous temperament; one of these had a macerated baby which she knew to be dead before coming into labour.

All the *secundiparæ* and *multiparæ* were delivered normally.

Of the *secundiparæ*, all but five considered the effect of the drug good. Two of the five, both exceedingly nervous, with big babies and fairly rapid labours, thought it fair. (Similarly, with the only two *multiparæ* who obtained merely fair relief, both were very nervous, with big children and very rapid and forceful contractions.) The three *secundiparæ* who found it unsatisfactory also had quick labours, of a total duration of six to eight hours. Although in these cases "Heptalgin" was given at the stage of three fingers' dilatation of the cervix, labour was probably advancing too strongly to be greatly affected, as delivery took place within less than two hours of the injection.

In no case was the duration of the effective dose longer than one hour and ten minutes.

Side-effects noted were vomiting in one case, transient giddiness in two, and a temperature of 101·6° F. with a rigor in one case. The woman who vomited consumed a full meal shortly after the injection, while the patient with the rigor, which took place half an hour after the injection, was admitted to hospital with pyelitis.

[1] Working under a grant from the King George V and Queen Mary Maternal and Infant Welfare Foundation.

TABLE I.

Group.	Age Range. (Years.)	Assessment.	"Heptalgin."	Pethidine *plus* Hyoscine.	Pethidine.	Potassium Bromide and Chloral Hydrate.
Total	18–41	Good	29	32	35	3
		Fair	5	7	3	24
		Unsatisfactory	6	1	2	13
Primiparæ	18–32	Good	10	12	14	1
		Fair	3	2	0	9
		Unsatisfactory	1	0	0	4
Secundiparæ ..	21–35	Good	8	10	11	1
		Fair	2	3	2	9
		Unsatisfactory	3	0	0	3
Multiparæ	21–41	Good	11	10	10	1
		Fair	0	2	1	6
		Unsatisfactory	2	1	2	6

Of those patients, 29 in all, who considered the effect of the drug good, 10 slept after injection, six were drowsy, and 13 rested quietly. None was amnesic.

The effect on the baby appeared to be minimal. All but two babies cried immediately on birth and were of good tone and colour. One baby of seven pounds eleven ounces was rather limp and cried after three minutes. It had fitted tightly in the perineum, which was preserved intact at the cost of considerable delay. The mother had experienced good relief in a ten-hour labour. The other baby was macerated.

Only two patients received a second dose, both *primiparæ* undergoing long labours (thirty-six and forty-five hours respectively), who were delivered instrumentally. The initial dose of 10 milligrammes at the stage of three fingers' dilatation was followed at the stage of four fingers' dilatation by one of five milligrammes. Both patients considered their analgesia good, and neither baby showed any ill-effects, either from the drug or from the long duration of labour.

The results gained with intramuscular injection of pethidine 100 milligrammes, and with intramuscular injection of pethidine 50 milligrammes *plus* hyoscine 1/300 grain, were very similar to those obtained with "Heptalgin" (Table I).

Several patients became restless and disorientated after the injection of pethidine *plus* hyoscine, but appeared to have no recollection of this later, although they remembered other incidents of the labour clearly, and the amnesia did not seem to relate to their pain.

Effects on the baby in all other series, including the series of patients who had no analgesia, were comparable with those from "Heptalgin".

The average duration of labour in the different series was as shown in Table II.

Clinically, the impression was gained that "Heptalgin" was in general a little more effective in relieving pain than 100 milligrammes of pethidine or 50 milligrammes of pethidine *plus* 1/300 grain of hyoscine, given intramuscularly, and considerably more so than potassium bromide and chloral hydrate given orally. It appeared to allow the cervix to dilate fully and the second stage to be entered more rapidly, whether the patient stated that she had experienced relief or not. In some, this rapid dilatation was felt to be responsible for the statement that the drug was ineffective.

Comment on Ten Other Patients.

Most of the 10 patients requiring separate discussion had prolonged labour, and all except one, who came to Cæsarean section, received doses of other analgesics in addition.

The patient who came to Cæsarean section had been labouring for fifty-six hours without advancing beyond three fingers' dilatation of the cervix, and without the administration of any analgesic, although she was of a highly nervous temperament; she was a primiparous ex-hospital sister, aged thirty-two years. She was exhausted and hysterical. One hour after the intramuscular injection of 10 milligrammes of "Heptalgin" her cervix was fully dilated and she was much calmer. After a trial labour of a further three hours, with inhalation of "Trilene", she was delivered of a live baby by Cæsarean section. She considered the "Heptalgin" better than the "Trilene", although the latter was not left to autoadministration.

TABLE II.
Duration of Labour (Hours).

Group.	"Heptalgin."	Pethidine *plus* Hyoscine.	Pethidine.	Potassium Bromide and Chloral Hydrate.	Nil.
Primiparæ ..	15·5	24	26	24	25·5
Secundiparæ ..	10	17	17	13	13
Multiparæ ..	13	9	14	9·5	13

Two patients were given by intramuscular injection 10 milligrammes of "Heptalgin" at the stage of three fingers' dilatation, after preliminary doses of one ounce of potassium bromide and chloral hydrate. Both found it infinitely superior.

A *secundipara*, given by intramuscular injection "Heptalgin" 10 milligrammes at the stage of three fingers' dilatation (whereafter she was drowsy) and also by intramuscular injection of pethidine 100 milligrammes at the stage of four fingers' dilatation, next day expressed the opinion that the first injection was more effective and that the whole labour had been much more comfortable than her first, when she had received no relief.

The remaining six patients were given by intramuscular injection pethidine 50 milligrammes *plus* hyoscine 1/300 grain, as well as "Heptalgin" 10 milligrammes. All had long labours (average thirty-two hours) and were young *primiparæ* aged nineteen to twenty-five years, except one very apprehensive *secundipara* aged thirty-three years. Four, including the *secundipara*, found its effect good, and superior to the pethidine and hyoscine. The other two experienced, or would admit, little relief from either.

Oral Use.

A number of patients were given "Heptalgin" by the oral route in a dosage of 10 milligrammes in tablet form. Relief obtained was nil, or very slight only. No vomiting was observed.

Summary.

"Heptalgin" was administered to 50 patients in labour. In the majority of cases it relieved pain to some extent without harmful side-effects to the mother or depressant effects to the baby. It appeared to shorten labour.

Forty patients so treated are compared with 40 patients given different analgesia, with the conclusion that "Hep-

talgin" is at least as good as pethidine, or pethidine *plus* hyoscine, in comparable doses, and better than potassium bromide and chloral hydrate given orally, or no analgesia whatever.

Acknowledgement.

I am indebted to the Glaxo Laboratories for supplies of "Heptalgin" for carrying out this experiment.

References.

Hewer, A. J. H., and Keele, C. A. (1948), "A Method of Testing Analgesics in Man", *The Lancet*, Volume II, page 683.

Wilson, W. M., and Hunter, R. B. (1948), "'C.B.11': A New Analgesic Drug", *British Medical Journal*, Volume II, page 553.

INTRAUTERINE FŒTAL ABNORMALITIES.[1]

By D. G. MAITLAND,

Sydney.

I HAVE selected for discussion 30 cases of intrauterine fœtal abnormalities of obstetrical interest. They are selected cases, which I have collected during my sixteen years' association with the X-ray department of the Women's Hospital, Crown Street, Sydney, and more recently with the department of the King George V Memorial Hospital for Mothers and Babies, Camperdown, New South Wales.

The X-ray diagnosis depends either upon the demonstration of actual skeletal abnormalities or the inference of an abnormality from an abnormal attitude of the fœtus in its relationship to the uterine shadow.

Skeletal abnormalities may be divided into gross and minor defects, but, in broad terms, the latter are of academic rather than obstetrical interest, because of the probable association of anomalies in other parts of the fœtus which cannot be discovered until after birth. I refer to the congenital cardiac and other lesions which may be associated with minor skeletal changes in the hands and feet.

It must be remembered, however, that gross fœtal abnormality may exist without any clinical evidence or suspicion of its presence, and it may be a wise precaution therefore to subject all candidates for Cæsarean section to a preliminary radiographic examination of the fœtus.

In this work the technical quality of the radiographs must be of the highest standard, and two or more views are necessary in order to exclude distortion as a possible factor in the production of abnormal shadow projections.

The type of X-ray equipment which is available in Australia varies considerably—particularly in country centres—yet excellent technical work can be obtained with an X-ray unit of modest output, provided certain physical principles are borne in mind and certain accessory appliances are available.

For radiography, the position of the mother should be that which demonstrates the fœtal shadows clearly and with the minimum enlargement or distortion.

I prefer the prone, semioblique and lateral views, for the physical reason that the object to be radiographed should be closest to the film. For the same reason, the focal spot of the X-ray tube should be the smallest practical size for sharp definition of the shadows.

High kilovoltage and low milliamperage work is preferable, because of the thickness of the tissues and density of the amniotic fluid to be penetrated by the X rays, and because, in general terms, "a rise of 10 K.V. halves the exposure time".

An old-fashioned, coarse, Potter-Bucky diaphragm is preferable to the more modern high-speed grid, because the latter has been designed for general radiography using low kilovoltage and high milliamperage techniques, which I have found unsuitable for use in general cases of pregnancy.

The "DuPont Hi-Speed" screens are extremely sensitive to the higher kilovoltage techniques, and these screens, together with the more speedy yet fine-grained films, have proved a distinct advantage in cases of pregnancy.

An adequate cone is essential in order to control scattered radiation and improve the quality of the radiograph.

I should first like to compare two films obtained in an average full-term pregnancy. The first was obtained in 1·25 seconds with a fine-focus stationary anode tube and only 30 milliamperes at 90 kilovolts peak. The second film was obtained with modern, high-powered hospital equipment in 0·5 second but with an indifferent technique. In this second case the fine-focus rotating anode tube was not rigid, and so it produced the shadow projection effect of a broad-focus tube. The thin high-speed grid was not effective in eliminating the high-frequency scattered radiation, and an over-sized cone was used, which increased the quantity of scattered radiation. Detail and contrast are therefore greatly impaired, a grey, "fogged" appearance being seen in the film. In general terms, milliamperage governs the quantity of radiation, not its quality; and since the time exposure must be short—partly in order to eliminate fœtal movement—kilovoltage is the preferable variant in cases of pregnancy in which repeated examination may be inadvisable.

The foregoing remarks and examples are intended to draw your attention to the importance of technique and the application of physical principles to this type of work, and the following examples will emphasize these points.

The radiographic diagnosis of early pregnancy depends upon the demonstration of the shadows of the minute ossific centres of the developing fœtal skeleton. Clinically, the "pelvic tumour" may be the size of a five and a half months' gestation, but it may be the enlarged uterus of a multiple pregnancy of only three months' gestation, an early pregnancy complicated by fibromyoma, or a single (or multiple) pregnancy with hydramnios. Therefore, beware of a diagnosis of "ovarian cyst" or "dermoid".

In multiple pregnancy, carefully observe the shadow of each fœtus to ascertain whether they are of normal size for the period of gestation, complete, fused or dead.

Films in my series illustrate acrania with a dead twin, fœtal death compared with head moulding, and twins fused at the pelvic girdle. In the latter case the two heads, four arms, a common pelvic girdle and two legs may be seen in the film.

Anencephaly is one of the more common abnormalities. It is usually suspected by the obstetrician, but may be mistaken for a breech presentation or twin pregnancy and discovered accidentally by the radiologist.

A breech presentation which has been converted to a vertex presentation and reverts to a breech should be viewed with suspicion by the obstetrician. In such cases radiographic investigation is warranted, as can be illustrated by a film showing congenital absence of forearms and legs.

In my experience iniencephaly is a rare condition. I have seen only three examples, the distinguishing features of which were "extension" of the hydrocephalic head with absence of any occipital calcification, cervical or cervicothoracic *spina bifida* and a shortened fœtal trunk.

There is a remarkable similarity between the shadow projection in two of my films, one of an iniencephalic monster and the other of a normal fœtus. In the latter film, however, the occipital bone and cervico-thoracic curve are intact, and in the lateral projection in the same case the fœtus can be seen to be a normal one in a somewhat pendulous uterus. Be wary, therefore, of shadow distortion or of expressing an opinion upon a single film.

[1] The substance of this paper was presented as a lecture-demonstration at the inaugural meeting of the College of Radiologists (Aust. and N.Z.) and was illustrated with X-ray films.

A brow or face presentation is not uncommon, and the cause may be suggested by the type of pelvic inlet through which the fœtal head is attempting to pass. I have no example of thyreoid tumour—which I believe to be extremely rare. Opisthotonos, on the other hand, may be produced by an almost complete absence of amniotic fluid, and the example that I have, showing the fœtus before and after delivery, illustrates the hyperextension of the head and pressure deformities of the head and limbs due to this cause. Delivery in this case was by Cæsarean section.

Hydrocephalus may be gross or mild, and it is often a very difficult matter indeed to detect the milder forms. Shadow enlargement of the fœtal head is a normal projection effect when a fœtus with breech presentation is radiographed in the supine position of the mother, and the fœtal skull is often elongated because of pressure of the *fundus uteri*.

Gross hydrocephalus, however, cannot be mistaken, and an example in my series shows a large, smooth, generally thinned cranial vault, with wide sutures. There is no visible spinal defect, and the fœtal body appears small in comparison with the large head.

Another film shows a hydrocephalic fœtus presenting by the breech with the duplicated, wavy shadows of the cranial bones typical of craniofenestria. There is gross *spina bifida* involving the lumbo-sacral region. The association of these three skeletal defects is not infrequent.

A third film illustrates a mild form of hydrocephalus, in which the membrane bones of the skull have a slightly less dense calcification than most for the stage of maturity. In consequence the sutures appear to be slightly wider than normal. By close observation of this film one can see a wavy contour of the frontal region indicating craniolacunia. There is no visible spinal defect.

In the following two cases the appearances strongly resemble one another, in that both fœtuses have densely calcified, and apparently enlarged, heads.

In the first case can be seen broad and slightly irregular vertebral bodies, which shorten the fœtal trunk. The film also illustrates the short and broadened limb bones typical of achondroplasia.

In the second case there is no variation from the normal in the vertebral bodies, limb bones or ribs, but the slightly increased size of the skull—and the density of the cranial bones—suggests an achondroplasic fœtus of milder form than in the previous case. This opinion was strengthened by further investigations after birth, but the possibility of Albers-Schönberg disease was considered at the time.

Another example shows angulation deformity of the long bones with decalcification in a fœtus, which is lying in an occipito-posterior position. This is an example of *osteogenesis imperfecta*. General decalcification of the fœtal skeleton, deformities of the ribs and limbs, and an irregular hydrocephalic cranium with craniofenestria may be noted. The general decalcification is interesting, because a case has been reported in the literature in which a diagnosis was made solely upon the recognition of the faint shadows of the petrous temporal bones—there being no other skeletal shadow visible within the uterine opacity.

Another series of films illustrate the condition known as *hydrops fœtalis*, which occasionally has been encountered in Rh-negative mothers, and which may well be an extremely rare condition in the future.

This condition is associated with hydramnios, and the fœtal attitude *in utero* is characteristic, owing to the œdematous tissues and ascites. The shadows of the fœtal skeleton assume a frog-like attitude, and the ribs are elevated and compressed. The fœtal soft tissues may be outlined against the more dense amniotic fluid, and the conclusive diagnostic point is the demonstration of the œdematous scalp surrounding the cranial bones. In favourable circumstances the œdematous limbs also may be outlined.

The prominence of the abdomen and elevation and crowding of the ribs are not of themselves sufficiently diagnostic of *hydrops fœtalis*, since these radiographic signs could also be produced by a renal or other congenital abdominal tumour.

I should like to conclude this discussion with mention of a case which taught me a salutary lesson and which will remind us all that the obvious finding is not always the most important feature of any case.

The patient was approaching term. Clinically, the fœtal head would not fit into the pelvic brim and she was sent to the radiographic department with the suggestion that disproportion was present.

Pelvimetry was carried out by the standard projections for this examination, and the films show that the adequate pelvis has a slight transverse contraction—with the fœtal head in an occipito-posterior position. This is quite a common finding in cases of "suspected disproportion".

The pelvic inlet and subpubic angle views can be discarded; but if one looks very closely at the lateral view of the pelvis, which includes the fœtal skull and the upper thoracic region of the fœtus, there can be seen in the right top corner of the film the shadow of the distal end of one femur superimposed over the upper part of the thorax of the fœtus. In the relatively long time-exposure necessary to obtain the lateral view, movement of the fœtal arms could have occurred; but by accepting the obvious I failed to appreciate that the shadow was not a humerus and to order an examination of the fœtus itself.

The obstetrician did not suspect any fœtal abnormality, but subsequently an infant was born with congenital absence of both arms.

CUTANEOUS MELANOMA.[1]

By DERICK YEATES,
Brisbane.

LAENNEC wrote the first treatise on melanoma in 1806. Since then it has provoked world-wide interest amongst pathologists and clinicians, and it has come to be regarded as one of the most dreaded and unpredictable of all tumours.

In order that there will be no confusion of nomenclature, the benign pigmented mole will be referred to as a pigmented nævus and the malignant tumour will be called a melanoma.

No age is exempt from melanoma, but it is rare before puberty, although it can be transmitted from the mother to the fœtus *in utero*.

It occurs with about equal frequency in either sex, and in over 50% of cases it develops in a preexisting pigmented nævus.

In cases in which melanoma supervenes on a pigmented nævus, there is often a history of mechanical injury or of incomplete surgical removal.

Melanomata may occur anywhere on the skin surface, but the commonest sites are the lower extremity, the trunk, the head and the neck.

Pigmented nævi are extremely common and there are few individuals who do not possess one or two moles. On the other hand, melanomata are comparatively rare, so that the chances against the nævus becoming malignant are many thousands to one.

Once malignant change does appear, it is not unusual for the tumour to metastasize widely and rapidly, so that diagnosis must be early and treatment radical.

Pathology.

In order to understand the origin of a melanoma it is essential to appreciate the histology of the pigmented nævus. Willis (1948) considers that the nævus is not a tumour but a cutaneous malformation which is usually congenital.

[1] Read at a meeting of the Section of Surgery, Australasian Medical Congress (British Medical Association), Seventh Session, Brisbane, May-June, 1950.

The following histological features of the nævus are worthy of notice: (i) The nævus is covered with epidermis which has irregular branched papillæ. (ii) The columnar basal cells of the epidermis are deeply pigmented. (iii) The basal cells, especially near the apex of the papilla, tend to separate and pass into the corium. (iv) These nævus cells are round or oval and have relatively small, darkly staining nuclei.

Histogenesis of Skin Melanomata.

Most cutaneous melanomata arise from preexisting nævi. Willis considers that whilst the structure of young pigmented nævi shows plainly the epithelial origin of the nævus cells, that of early malignant melanomata shows equally plainly their origin from nævus cells and from the epidermis itself.

Willis does not believe that melanomata arise from cutaneous nerve endings.

Bloch's "dopa" reaction is a test for the presence of intracellular melanogen oxidase; the reaction is given in common by the basal cells of the epidermis, the cells of the hair follicles, nævus cells and malignant melanoma cells. It is not given by cells of mesodermal origin—for example, the melanophores.

Structure of Skin Melanomata.

The structure of skin melanomata is varied. Cells may be polyhedral, fusiform, spherical or pleomorphic. They may be arranged in epithelium-like clumps or in perivascular mantles or diffusely.

The cells may be deeply pigmented, but sometimes they contain no pigment.

Great experience is necessary in the microscopic diagnosis of early malignant changes in pigmented nævi. Features arousing suspicion are increase in size of the nuclei and cytoplasm of the nævus cells, cellular pleomorphism, the presence of mitotic figures, the invasion of the overlying epidermis by groups of atypical cells, and unequal distribution of intracellular pigment in different parts of suspected areas.

Spread of Melanoma.

The behaviour of no other tumour is so unpredictable as that of melanoma. A small, innocent-looking pigmented tumour may prove rapidly fatal from widespread metastases, whereas occasionally a frankly malignant melanoma is cured by simple excision.

The mode of spread of a particular melanoma is equally difficult to predict. Some tumours spread out in the superficial lymphatics by a process of lymph permeation. Others show little local growth, but spread in the deep lymphatic trunks by lymph embolism to regional lymph glands. Others disseminate solely by the blood-stream.

Regional lymph node involvement is common, and often it has already occurred before the glands are enlarged. This early involvement of lymph nodes commonly occurs by lymph embolism, and this fact has an important bearing on surgical treatment.

Treatment.

Pigmented Nævi.

Pigmented nævi are very common and often multiple, and generally speaking they should not be removed. However, if they are situated in such an anatomical position that they are constantly subjected to irritation—for example, on the collar line of the neck—then there should be no hesitation in advising wide excision under general anæsthesia.

Pack and Livingston (1940) consider that the common belief that removal of nævi may lead to the development of malignant disease is erroneous; the explanation is that the patient possessing the nævus has been motivated to have it removed because of some change which has developed in it—for example, increase in size, elevation of the nævus, hæmorrhage, ulceration, or other changes which called the patient's attention to this abnormality. These changes usually indicate that malignant change has already occurred, and local excision of the tumour is then unlikely to eradicate the disease. Pack does not know of a single case of simple pigmented nævus in which adequate excision has precipitated malignant change.

If excision is undertaken, it should be under general anæsthesia, and the area excised should be well clear of the nævus in all directions and should include all the tissues down to the muscle layer. Naturally such a specimen should be subjected to careful microscopic examination. If there is no sign of malignant change, then the above treatment is adequate and further trouble is unlikely. On the other hand, if the tumour is reported to be a malignant melanoma then it must be treated as such (see later).

Malignant Melanoma.

The primary tumour should be widely and deeply excised under general anæsthesia. If feasible, the regional lymph nodes are excised *en bloc* about two weeks later. This delay gives time to have the diagnosis confirmed microscopically, and also allows time for any particles of growth already in transit to lymph nodes to reach their destination by a process of lymph embolism.

If the excised lymph nodes show evidence of metastatic involvement then block dissection of a more proximal group should be considered; for example, in Case I the inguinal glands were grossly involved, so the deep iliac glands were excised *en bloc*.

X-ray therapy has proved of little value in the curative treatment of melanoma, but it is of occasional use in palliative treatment of inoperable melanoma.

Because of the occasional dissemination of melanoma via the lymphatics of the skin by permeation, some authorities have recommended that these tumours should be excised *en bloc* with the skin overlying the lymph vessels between the primary melanoma and the regional lymph nodes. Ackerman and del Regato (1947) consider that this radical procedure is unjustifiable, because most malignant melanomata metastasize by embolism through the deep lymph trunks rather than by permeation of the superficial lymphatics. Ackerman and del Regato further consider that when the tumour is situated in an area from which the lymph drainage is predictable, then a radical dissection of the anticipated metastatic node areas is mandatory in spite of the fact that the nodes show no clinical signs of involvement. The only exception to this rule may be the presence of a melanoma in the mid-line of the face, chest or back, for which a bilateral block dissection would have to be carried out. In these cases it would seem justifiable to await the possible clinical enlargement of glands.

Results.

My own cases of melanoma of the skin have been recorded since January, 1946. Of these patients, 14 have been subjected to radical block dissection of the regional lymph nodes according to the principles of treatment already laid down. Table I gives a summary of these dissections.

In three cases double dissections were indicated, the combinations being axillary and cervical, bilateral axillary and inguinal and iliac.

There were no operative deaths, but two patients (Cases VI and X) died subsequently from widespread metastases.

On the other hand, one patient is alive and well four years after block dissection of the inguinal glands following excision of a malignant melanoma of the foot. The inguinal glands were deeply pigmented and microscopic examination revealed obvious secondary deposits of melanoma.

Two other patients who had secondary involvement of lymph nodes show no sign of recurrence three years after radical block dissections.

In the remaining cases the patients are progressing satisfactorily, but this has been too recent to give any idea of the ultimate prognosis.

TABLE I.

Lymph Glands Removed.	Number of Block Dissections.	Number of Patients with Metastatic Involvement of Glands.
Inguinal	6	4
Axillary	8	2
Cervical	2	1
Deep iliac	1	Nil
Total	17	7

Conclusions.

1. There should be no hesitation in excising pigmented nævi which are subjected to chronic irritation. If this excision is correctly carried out it will not precipitate malignant change.

2. At the least clinical suspicion of malignant change radical excision of the primary growth is indicated.

3. Unless there is some strong contraindication, prophylactic block dissection of the lymph glands draining the area of the primary malignant tumour is advised.

4. No time should be wasted on X-ray and radium therapy as practically all melanomata are radio-resistant.

5. Most people possess one pigmented cutaneous nævus. or more whilst malignant tumours are comparatively rare. Consequently the great majority of patients presenting with a pigmented lesion of the skin will require no further treatment than reassurance.

Acknowledgements.

For their helpful advice in the preparation of this paper I should like to thank Dr. A. G. S. Cooper of the Queensland Radium Clinic, and Dr. D. G. Neill of the Department of Pathology, University of Queensland.

Bibliography.

Ackerman, L. V., and del Regato, J. A. (1947), "Cancer: Diagnosis, Prognosis and Treatment".
Pack, G. T., and Livingston, E. M. (1940), "Treatment of Cancer and Allied Diseases".
Willis, R. A. (1948), "Pathology of Tumours".

A SIMPLE CHIN SUPPORT FOR ANÆSTHESIA.

By A. P. BALTHASAR, D.A.,
Sydney.

THE device to be described has proved of value in anæsthesia for long orthopædic procedures at Saint Vincent's Hospital and the Royal South Sydney Hospital. It frees the anæsthetist's hands from the purely mechanical act of supporting the jaw. He may then attend to important functions, such as blood transfusion, blood pressure recording, adjustment of gas mixture *et cetera*. Thereby the inevitable petrification that gradually seeps from the anæsthetist's hand to his brain is prevented.

Nothing more formidable is required than an inextensible bandage about eight feet long and two inches wide, and a small strip of sticking plaster. The middle of the bandage is passed under the chin and immobilized there by the strip of sticking plaster which is affixed from the centre of the mask over the bandage and down onto the skin of the mid-line of the neck as far as the Adam's apple. The bandage is then passed on either side of the face and

FIGURE I.

looped over itself to form a slip-knot lying on the forehead. The two ends are taken vertically downwards over the head of the operating table and secured to any convenient anchoring point underneath. When tension is applied to the ends of the bandage the head and jaw will be caused to rotate about a fulcrum in the region of the occiput. The point of the chin is thus carried forwards and upwards with immediate freeing of the airway. The strip of sticking plaster not only immobilizes the bandage on the chin, but also keps the lower part of the inflatable rim of the mask in good apposition with the lower jaw. This is of some importance, as the chins of elderly, edentulous people invariably sag away from the mask, with resultant loss of the anæsthetic gases and the anæsthetist's temper.

It is essential that the slip-knot be placed on the forehead and not further back on the head, as only in this position does it serve to change the line of action of the pull, shown by the arrows, through a right angle. Thus the applied tension is downwards and backwards, but the resultant pull on the chin is upwards and forwards.

The bandage may be equally well used under an open mask.

Reports of Cases.

THE SYNDROME OF LAMELLAR CEREBELLAR DEGENERATION ASSOCIATED WITH RETINITIS PIGMENTOSA, HETEROTOPIAS, AND MENTAL DEFICIENCY, WITH REPORT OF A CASE.

By P. B. HAGEN, K. B. NOAD AND OLIVER LATHAM,
From the New South Wales State Laboratory of Mental Hospitals, Department of Pathology, University of Sydney.

THERE is a tendency today to link together in a syndrome various degenerations in different parts of the body, sometimes associated with some developmental stigmata. One of these syndromes which has come to our notice we wish to report. The patient was an aged man who had never

been to school, but who was astute, law-abiding and agreeable, and with a little help looked after himself for most of his life. Alive, he exhibited cataracts and certain ataxic features; after death, a small cerebellum, the seat of most severe chronic parenchymatous cortical cerebellar atrophy, gross degeneration of his granule cells, grave myelin deficiency, remarkable glial hypertrophy, and innumerable and rare intrusion neurons of strange aspect and peculiar situation—heterotopias.

Clinical Record.

E.C.K. was admitted to the Lidcombe State Hospital in 1944 at the age of sixty-nine years, with a diagnosis of locomotor ataxia. He failed to recognize anything wrong with himself. He had had no schooling and could never read or write. He denied previous illnesses.

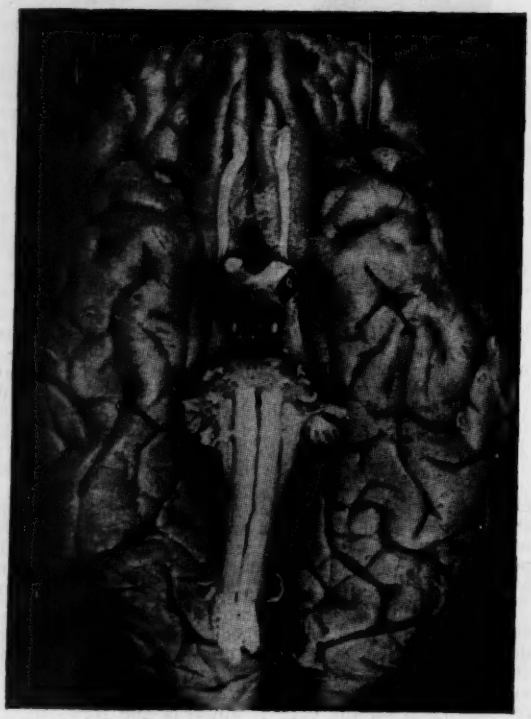


FIGURE I.

W. Freeman's case. Photograph of under-surface of the brain, showing minute cerebellum, poorly developed medulla and the usual deficient development of the basilar pons (pontine nuclei and peduncle *et cetera*); yet the patient lacked any motor inconvenience till late in life.

Physical examination at that time revealed him to be a man of subnormal intelligence. His speech was suggestive of cerebellar disorder. Scoliosis was present, and bilateral inguinal herniæ were controlled by a truss. His gait was moderately ataxic, and slightly irregular choreiform movements were obvious in the upper limbs. No record of visual acuity was made. The pupils were slightly irregular, but reacted to light. There was paralysis of the lateral rectus on one side. An intention tremor was present, but no other cerebellar phenomena were noted in the upper limbs. The deep reflexes were exaggerated. A lumbar puncture was performed with the subject in the sitting position, and a pressure of 245 millimetres was noted. The cerebro-spinal fluid contained no cells, 40 milligrammes of protein per 100 millilitres, and 740 milligrammes of chlorides per 100 millilitres. The colloidal gold curve was represented by the following figures: 1112100000. The Wassermann and Kahn tests both produced negative results in both blood and cerebro-spinal fluid.

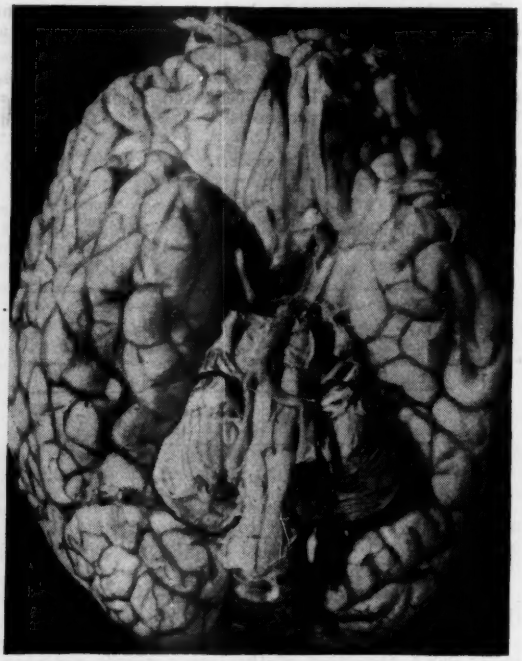


FIGURE II.

Our case; similar photograph. The cerebellum, though much larger, is still very small. However, the pons and medulla are not unusually small for an aged man. The outline of the cerebellum has been inked in.

FIGURE III.

Transverse section of half the medulla. The myelin stain shows normal myelin save for the circumolivary fibres and those proceeding from the hilum and crossing the accessory olive, which are diminished, although not to the extent one would have expected from the great loss of olivary nerve cells and from the extent of the substitution gliosis.

In 1946, aged seventy-one years, the patient was re-examined. He still could give no adequate history. He mentioned a "fit" at five years, and dated the onset of his speech difficulties and locomotor disorder to his eighth year. His brother's wife, the only relative available, said she was told that he had been born with some nervous disorder of which she knew no details. His parents, a brother and two sisters were all dead, and she was certain that they had died of complaints quite unlike that from which the patient was suffering.

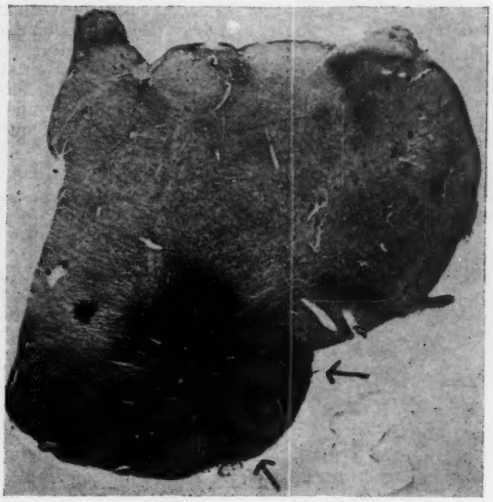


FIGURE IV.

Consecutive section to that shown in Figure III, only stained by Mallory's phosphotungstic hæmatoxylin. The stain shows the whole olive as a woolly mass of purple on a red medulla. The glial fibres overspread everything in the olive and come out dark in the photograph.

On examination in 1946 he was ataxic and could not walk without assistance. Articulation was explosive and the voice monotonous. Bilateral immature cataracts were present, which obscured the fundi. Perception of light only was noted. Internal strabismus was present on both sides, but otherwise the cranial nerves were intact. Spontaneous

FIGURE V.

Portion of an internal folium. The pia is thinner, yet only nuclei represent the remains of Purkinje cells (shown by arrows), and the molecular layer is traversed by vertical stripes—that is, proliferated Bergmann glia. "G" and "H" and other blobs are Golgi neurons *et cetera* in the granular layer, which is almost devoid of granule cells.

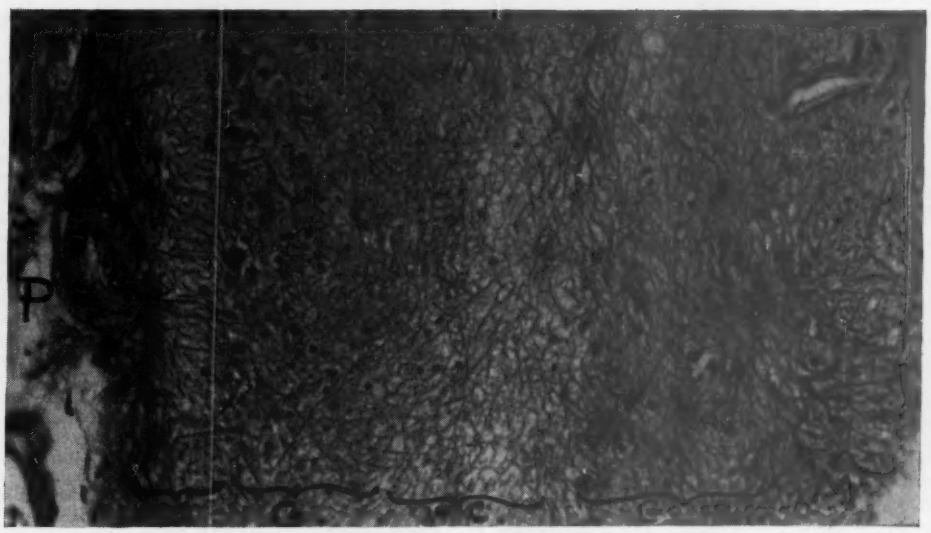


FIGURE VI.

Transverse section right through part of a folium (external surface) with constituent layers, molecular (M), granular (G) and white centre (W-C) indicated. The last-mentioned is devoid of myelinated fibres, being represented by tangled and cystic masses of glial fibres and cells. The granular layer is without granules, while the absence of Purkinje cells and the presence of the Bergmann glia and thickened pia-arachnoid are evident at P.

movements were absent, but some suggestion of cog-wheel rigidity was present in the upper limbs. There were also past pointing and some ataxia in the finger-nose test. The deep reflexes of the arms were present and equal, and sensation was normal in all modalities. Abdominal reflexes could not be elicited and the cremasteric reflexes were diminished. The knee and ankle jerks were brisk and equal and the plantar reflexes were flexor. The cerebrospinal fluid pressure was 280 millimetres with the patient in the sitting position; the protein content was not increased; the Wassermann and Kahn tests failed to produce reactions, and no curve was obtained with gold sol.

To recapitulate, the salient features displayed by this patient were as follows: (i) ataxia of speech, arms and legs; (ii) bilateral abducens palsy; (iii) absent abdominal reflexes; (iv) brisk knee and ankle reflexes with flexor plantar responses; (v) blindness.

The patient continued his semi-vegetative existence till April 5, 1948, when he began to vomit. This he did for several days, and then settled down until May 6, when vomiting recommenced and continued for a week. The temperature rose and the patient became very toxæmic. Two days later he died.

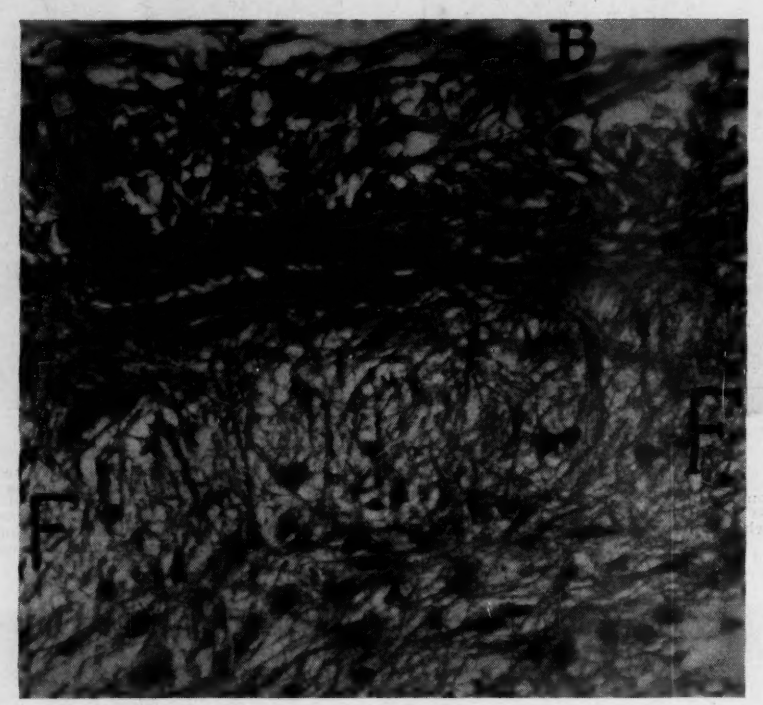


FIGURE VII.

Molecular and superficial part of the granular layer of previous section highly magnified. The tiers of proliferated Bergmann glial nuclei and coarse fibres (F-F) pierce the thickened pia between B and B, forming so-called brushes on the surface.

Post-Mortem Report.

At autopsy a few hours after death a ruptured appendix was found, and a thick-walled abscess which had burst, giving rise to generalized peritonitis.

The pia-arachnoid covering the cerebral hemispheres and cerebellum was tough and thick.

On removal of the cranial contents, it was seen that the posterior fossa and the cerebellum were both very small, the cerebellum failing to reach the borders of the occipital lobes. The medulla, the pons, and the cerebral and cerebellar peduncles were all diminished in size and the olivary eminences inconspicuous.

The brain weighed 1196·74 grammes and, this being taken as 100%, the hemispheres weighed 1131·0 grammes or 94·5% of the total, the cerebellum weighed 46·5 grammes or 3·8% of the total, and the pons and medulla weighed 19·25 grammes or 1·7% of the total. The small size of the cerebellum can be appreciated when it is realized that it was only 3·8% of the total brain weight as compared with the normal 11%—that is, approximately 150 grammes.

The right cerebral hemisphere was sliced, and after formalin fixation put through gelatin.

Paraffin sections were made from both hemispheres in the following regions: frontal, ascending frontal, parietal, temporal, occipital and hippocampal; from the posterior and inferior horns of the lateral ventricle and from the caudate nucleus, striate body, island of Reil, optic thalamus and *corpora mammillaria*.

Frozen sections were made from the cerebral cortex and peduncles, two levels of the pons and medulla, four levels of the cord and three parts of the cerebellum.

A complete antero-posterior section was taken according to the method of Tilney and Riley through the vermis and one cerebellar hemisphere, and from the other hemisphere a similar section further out to include the dentate nucleus and certain roof nuclei.

Stains and impregnations used included hæmatoxylin and eosin, Nissl's, the Mallory stains, a quadruple stain, Weil's stain for myelin, 20% alcoholic toluidin blue for frozen sections, *Scharlach R* and Gibson's modification of Cajal's stain for axis cylinders and cerebellar neurons, von Braunmuhl's stain, cresyl violet *et cetera*, as well as the King Anderson and Cobb Pilcher silver techniques.

While in the neurons of many areas in the cerebral hemispheres were found spindling on a vascular basis and axonal degeneration, much possibly reversible, in other areas there were numerous large neurons filled with pigment, some frankly degenerate, and others had disappeared entirely. However, there were innumerable large neurons, such as the Betz cells, which were in an excellent state of preservation connoting good fixation.

The neurons of the second and third layers had suffered most. This general description can be applied to all the areas examined down to the medulla and cord.

Gliosis was patchy, many areas in the temporal lobes were particularly gliosed and contained amyloid bodies, and argentophile plaques were seen in every area examined, but not nearly to the extent of an average case of Alzheimer's disease. The ground substance was deficient in patches of a similar size, and these areas were unstainable by any method—all this denoting some degenerative phenomenon.

The blood vessels require comment. The larger arteries were not much affected, but a few of the larger arterioles in the striatum were ferruginized, while in parts of all areas examined the condition of the arterioles varied between productive arteritis and fibro-hyaline degeneration.

Both eyes and their optic nerves were included in the examination.

The optic nerves seemed to contain a normal number of myelinated fibres and axis cylinders, as did the nerves to the extrinsic muscles. But on sectioning the globes, Dr.

V. J. McGovern, pathologist of the Post-Graduate Committee in Medicine, University of Sydney, readily pointed out the presence of *retinitis pigmentosa*, and examination of the

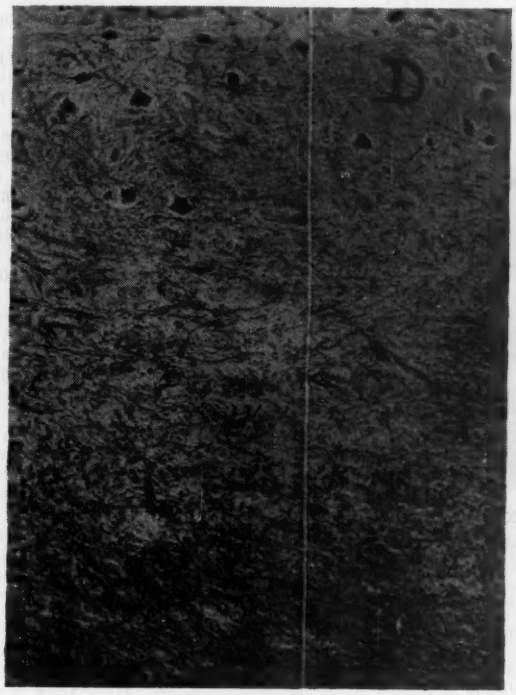


FIGURE VIII.

Neurons of the dentate nucleus are normal (D), but the surrounding myelin fibres forming the capsule and those entering the hilum are thinned. Near the folia myelin quite disappears. However, a thick band of myelin (at the bottom of the photograph) separated the dentate nucleus from the ventricle. Nerve cells from nearby nuclei also are normal. (Weigert-Pal stain.)

paraffin sections revealed severe retinal degeneration extending from the periphery inwards and most pronounced in the middle zone, where the various retinal layers were destroyed as well as the ganglion cells. In parts the condition seemed mildly cystic. Calcareous particles were also found in the lenses, with the production of a degree of cataract.

The pontine nuclei and tracts were well preserved and those of the arcuate nuclei as well. In the medulla, however, while the various tracts and neuron groups were well preserved, there was extreme atrophy of all the inferior olives, only a few scattered nuclei of the olivary neurons remaining. The myelinated fibres of the tuber regions were grossly degenerated, more so than the axis cylinders. The whole olive system, then, was the site of a dense glial cell and fibre reaction.

The Cerebellum.—The cerebellar peduncles, while rather small, were well myelinated, though in one part of the inferior or restiform body some myelinated fibres appeared to be missing, and in other parts swelling and beading of the fibres were apparent. The folia of the cerebellum generally showed a tendency to be lance-shaped. On microscopic examination the molecular layer was extremely thin and the layer of granules almost non-existent. The absence of myelinated fibres was almost complete, at least in the folia themselves. These appearances were seen in almost every folium. Not one rounded normal Purkinje neuron was encountered, and very few were found with any process save a few revealed by silver methods. No torpedoes could be recognized. The Bergmann glia cells were proliferated till they came to be arranged in many rows, while their processes, mingling with those of the superficial pia, formed such masses of tangled strands as to produce "bushes" which in many places burst through the thickened pia,

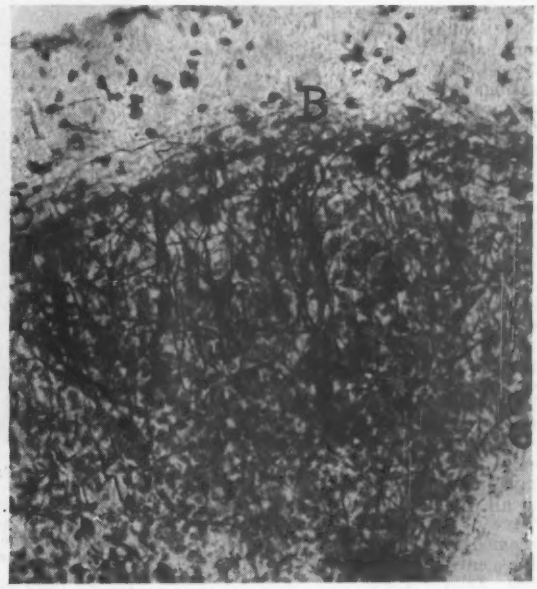


FIGURE IX.

Silver preparation of a folium, revealing horizontal fibres and the enormous long, thick and bushy basket processes, such as birds have. Much larger ones defied photography. Dark spots in the granular layer (as G) are Golgi type II cells. Note the absence of Purkinje cells, usually well shown by this method.

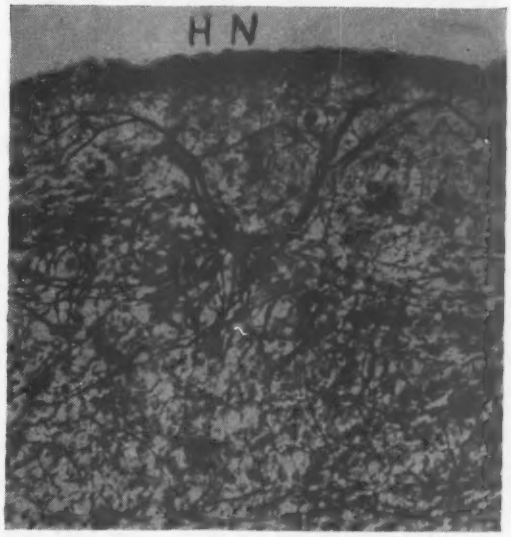


FIGURE X.

Untouched photograph of a similar silver reaction, showing gigantic abnormal nerve cell under HN in the position of the Purkinje cells, but with widely branching dendrites or plumes spreading far beyond the confines of the section. In the bottom right corner axis cylinders appear plentiful, but most of the twisted fibres are glial.

forming excrescences on the surface. The granular layer had for the most part quite disappeared, the many nuclei seen belonging obviously to glial cells. In this layer an extraordinary number of neurons were found—possibly Golgi type II cells, but at times with rather too long processes. A number of similar enlarged neurons were found in the molecular layer. Now Cajal preparations and Golgi methods depict in the normal molecular layer peculiar elongated neurons called stellate cells. Enormous cells resembling these were found in this case. The extreme degeneration of the myelin and the dense profusion of glial cells and fibres led to great staining difficulties, which, when overcome, revealed no myelin structure at all in the folia. The myelin seemed to stop at the entrance to the folia. The dentate nucleus and possibly the *nucleus globosus* were well preserved, only a few cells were missing, and the myelin in and around the hila of these structures, though thinned, was evident.

FIGURE XI.

Further thick frozen silver section, showing in the molecular layer a typically pyramid-shaped neuron of cerebral type lying horizontally and parallel to the horizontal fibres. It has an axon hillock (cut through by the section mounting needle) and the usual dendrites, one of which curves back like those of stellate cells. The body and recurved process have been outlined in ink, as has also been a ganglion cell N in the granular layer, which shows many more such cells. Rather more granule cells are visible.

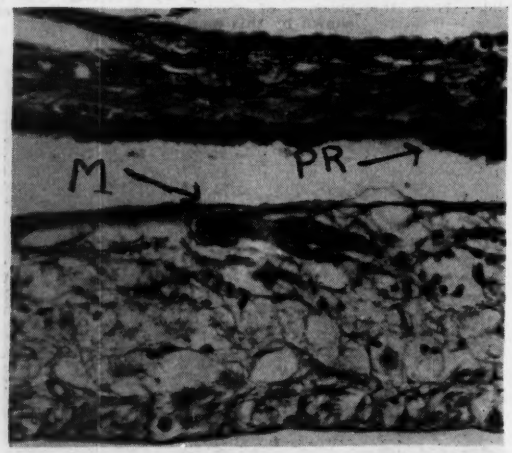


FIGURE XII.

Section of the retina. The pigment layer of the retina (PR) is clearly hypertrophied, and the masses of pigment in it are obvious, as at PR. A further mass is seen in the retina further in at M. The retina is severely atrophied; there are deficiency of nerve fibre layer, absence of ganglion cells and the deep granular layer, and at this point no rods and cones. These make their appearance for a few millimetres more peripherally. The general picture suggests a cystic condition. The optic nerves were normal.

Discussion.

This patient appears to have suffered from chronic parenchymatous cortical cerebellar atrophy with accompanying degeneration of olivary neurons and a profuse glial overgrowth. However, some might follow Kinnier Wilson's classification and on account of the gross neuron degeneration and glial overgrowth in the olives, plump for the olivocerebellar type.

When the small cerebellum was found at autopsy, a developmental anomaly was considered to be the probable basis of the condition. However, the discovery of *retinitis pigmentosa* put a different complexion upon the case and at once suggested a degenerative disease of the nervous system. It is at times extremely difficult to decide, except with the microscope, whether atrophy of the cerebellum and its processes is aplasia, or hypoplasia, or a true atrophic (that is, degenerative) process. Gordon Holmes states that when neurons are found to be absent, especially if their remains persist, and dense gliosis is found, a degenerative process rather than a congenital defect is the underlying cause. Sufficient olivary and Purkinje squibs remain here as evidence that these cells once existed. The preservation of the dentate and other nuclei and of the cerebellar peduncles is in accord with the idea of a degenerative syndrome, as congenital absence of the cerebellar cortex may lead to transneuronal degenerative changes in corresponding portions of these central nuclei.

Mere smallness of the mid-brain and cerebellum is not necessarily indicative of a congenital malformation, as we are constantly meeting in this laboratory small structures, similar to those present in this case, in such diverse conditions as cerebral arteriosclerosis, Huntington's chorea, and cerebellar degenerations generally. As structures may increase in size from additions of adventitious cells, as in inflammatory conditions, so they may diminish from the disappearance of neurons and their processes.

It is probable, however, that in this case a degenerative process was superimposed on a developmental background, as both the patient and one of his relatives related the onset of his symptoms to a very early age, and evidences of immaturity were to be found in the central nervous system, such as an increase in the number and size of neurons in both the granular and molecular layers and many nerve cells with double nuclei.

Other cases such as this have been described. Duke-Elder records the frequent combination of optic atrophy or *retinitis pigmentosa* with other central nervous system syndromes. R. M. Stewart (1937) has described two sisters with cerebellar ataxia, mental deficiency and *retinitis pigmentosa*. Kinnier Wilson also mentions optic atrophy and monoplegias among his hereditary cerebellar ataxias.

Froment, Bonnet and Cobrat have studied a family in which both cerebellar ataxia and spastic paraplegia have developed in association with pigmentary degeneration of the retina in several generations. Zonca, too, found this condition in association with Friedreich's ataxia.

Freeman's case was of great interest, in that the only parts of the cerebellum developed probably represented those earliest derived from the rhombic lip in connexion with the vestibular system. Also he stresses that some mental deficiency is usually present in these cases, and quotes others who maintain that complete absence of cerebellar tissue must give rise to some physical signs. Another point he alludes to is that severe cerebellar deficiency is

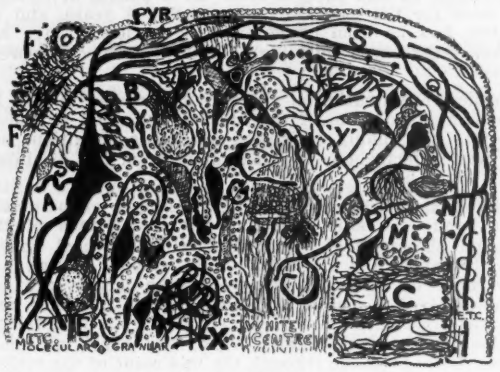


FIGURE XIII.

Diagram of the heterotopias. This diagram illustrates structures, chiefly neurons, not lending themselves to photography. Structures stained black with silver are thus rendered here, while those staining only brown or yellow are in stipple. Note the thick processes (of abnormal neurons) in the white centre, some from Golgi type II cells or else ganglion cells in the granular layer *et cetera*, whose dendrites try to seek the molecular layer or form horizontal fibres or tufts under the Purkinje cells. At X is a whorl of such fibres, but rather too large perhaps for a cerebellar glomerulus. V, E, A, B, Y, "Y", P and M are abnormal cells, and B has a thick process; all layers hold many such. A, possibly a gigantic Purkinje cell, has processes exceeding 1000μ in length; however, no healthy Purkinje cells or torpedoes were noted. F and "F" are Bergmann glial brushes on the pial surface; under Q are nerve cells upside down, and the normal artist brush-like basket as at N is here at C long, thick and bushy. The arrow at K points to degenerated Purkinje nuclei. Now Golgi type II neurons in the granular layer have been supposed to have their axon end by at once breaking up into local brushes. Cajal more recently has revised this opinion by stating that some must be designated "ganglion" cells and send their axon to leave the cerebellum by some peduncle. Many examples of this phenomenon can be noted—for example, at Y and "Y" in this diagram.

associated with under-development of the basilar pons only, the pyramids being thus allowed to protrude on its ventral surface. Apart from no cerebellum at all, Freeman seems to have presented quite the smallest of which we know. The cerebellum was represented by two nubbins of cerebellar tissue and no fourth ventricle existed, a membrane covering the area. Yet no symptoms attributable to such an agenesis showed up in life till nearly seventy years had passed, when arteriosclerosis removed cerebral help, on which cerebellar signs manifested themselves. Freeman quotes another case in which cerebral syphilis similarly unmasked a cerebellar abnormality or agenesis. Freeman's patient was always subnormal and had had no schooling, but walked and talked well at the usual age, supported himself at gardening and carpentering, and could count money.

Summary.

1. A case has been presented embodying mental deficiency and *retinitis pigmentosa*, with the accompanying defects of severe retinal degeneration and cataracts. Also present were chronic lamellar cerebellar degeneration, grave loss of granule cells and myelin, extreme gliosis and secondary degeneration of the medullary olives as well as remarkable and rare heterotopias.

2. Similar cases of other authors have been mentioned.

3. Some notes have been embodied discussing to what extent the small size of these cerebelli depend on slow parenchymatous degeneration and sclerosis and on the original developmental accompaniments.

4. The curious fact emerges that almost complete absence of the cerebellum seems to be accompanied by fewer clinical phenomena than the moderate degeneration of a fairly normal cerebellum.

Acknowledgements.

The patient was under the care of one of us (P.B.H.), who prepared many of the sections in association with Dr. W. C. Gibson. Acknowledgements must be made to Dr. E. S. Morris, Inspector-General of Mental Hospitals, New South Wales, and Dr. Grey Ewan, Director of Mental Hygiene, for permission to publish this case; to Professor W. K. Inglis and Professor A. N. Burkitt for their continued help and interest; to Miss Hazel Hunter, librarian, for looking up many references; to Mr. Woodward-Smith and Mr. Ken Clifford, of the Department of Medical Artistry, University of Sydney, for the illustrations; and to Mr. Tom Jones for some of the sections.

References.

Abbie, A. A. (1941), "The Anatomy of the Cerebellum", THE MEDICAL JOURNAL OF AUSTRALIA, Volume I, page 159.

Aring, C. D. (1940), "Degeneration of the Basal Ganglia Associated with Olivo-Ponto-Cerebellar Atrophy", *The Journal of Nervous and Mental Disease*, Volume XCII, page 448.

Biggart, J. H. (1948), "Pathology of the Nervous System", Second Edition, Edinburgh, E. and S. Livingstone.

Brain, R. (1933), "Diseases of the Nervous System", London, Humphrey Milford.

Brushfield, T., and Wyatt, W. (1927), "Microcephaly", *British Journal of Children's Diseases*, Volume XXIV, page 24; quoted in *British Medical Journal* (1927), Volume I, page 117.

Cajal, R. (1933), "Histology", translated into English from the Tenth Spanish Edition, London, Baillière, Tindall and Cox.

Campbell, A. W., Lichfield, W. F., and Latham, O. (1917), "A Clinical and Anatomical Report of a Case of Friedreich's Ataxia", THE MEDICAL JOURNAL OF AUSTRALIA, Volume I, page 136.

Cooper, E. R. A. (1946), "Development of the Substantia Nigra, Red Nucleus and Corpus Striatum", *Brain*, Volume LXIX, Part I, page 22.

Courville, C. B. (1949), "Pathology of the Central Nervous System", Second Edition, California, Pacific Press Publishing Association.

Donaldson, H. H. (1895); "The Growth of the Brain", London, Walter Scott, Limited.

Duke-Elder, W. S. (1945), "Text Book of Ophthalmology", Volume III, page 276.

Ford, F. R. (1946), "Diseases of the Nervous System in Infancy, Childhood and Adolescence", Springfield, Charles C. Thomas.

Freeman, W. (1933), "Neuropathology: The Anatomical Foundation of Nervous Diseases", Philadelphia, W. B. Saunders Company.

Gibson, W. C. (1937), "Silver Methods for Boutons Terminaux and Neurofibril. McClung's Microscopic Technique", Second Edition, New York, Paul B. Hoeber.

Hall, B., Noad, K. B., and Latham, O. (1945), "Familial Cortical Cerebellar Atrophy: A Contribution to the Study of Heredo-Familial Cerebellar Disease in Australia", THE MEDICAL JOURNAL OF AUSTRALIA, Volume I, page 101.

——— (1941), "Familial Cortical Cerebellar Atrophy", *Brain*, Volume LXIV, page 178.

Lambie, C. G., Latham, O., and McDonald, G. L. (1947), "Olivo-Ponto-Cerebellar Atrophy", THE MEDICAL JOURNAL OF AUSTRALIA, Volume II, page 626.

Lichtenstein, B. W. (1941), "Maldevelopments of the Cerebellum", *The Journal of Neuropathology and Experimental Neurology*, Volume II, page 164.

Minogue, S. J., and Latham, O. (1945), "Cerebellar Degeneration with Epilepsy", THE MEDICAL JOURNAL OF AUSTRALIA, Volume I, page 430.

Mollendorff, W. S. (1928), *"Handbuch der mikroskopischen Anatomie des Menschen"*, Berlin, Julius Springer.

Rubenstein, H. S., and Freeman, W. (1940), "Cerebellar Agenesis", *The Journal of Nervous and Mental Disease*, Volume XCII, page 489.

Stewart, R. M. (1937), *Proceedings of the Royal Society of Medicine*, Volume XXX, page 849.

Tilney, F., and Riley, H. A. (1921), "The Forms and Functions of the Central Nervous System", Paul B. Hoeber.

Van Leeuwen, M. A., and Van Bogaert, L. (1949), "Hereditary Ataxia with Optic Atrophy of the Retro Bulbar Neuritis Type and Latent Pallido-Luysian Degeneration", *Brain*, Volume LXXII, Part III, page 340.

Wertham, F. and F. (1934), "The Brain as an Organ", New York, The Macmillan Company.

Wilson, S. A. Kinnier (1940), "Neurology", London, Edward Arnold and Company.

ARTERIOSCLEROSIS OBLITERANS INVOLVING THE UPPER LIMBS: CASE REPORT.

By PHILIP RUNDLE,
Newcastle.

THE occurrence of degenerative vascular disease involving the limb vessels has been the subject of much clinical interest and investigation in recent years. General knowledge of the clinical manifestations of this process has increased with the advent of newer medical and surgical methods of treatment which have, at least in many cases, resulted in the delay in occurrence of incapacitating sequelæ of tissue destruction, although certain cure of the basic vascular lesion is as yet unknown. It is, however, unfortunate that the essential factors concerned in the evolution of the condition are still poorly understood and there is no universal acceptance of the cause of *arteriosclerosis obliterans*.

Symptoms due to *arteriosclerosis obliterans* usually make their appearance in the decline of life, being most common after the fifth decade. They generally occur first in the distal vessels of the lower limbs, giving rise to a well-recognized picture of intermittent or persistent pain and colour or temperature changes in the skin, both of an objective and a subjective nature; later trophic changes appear as the nutrition of the tissues is affected. This occurrence, giving rise to symptoms primarily due to involvement of the distal vessels of the upper limbs without other evidence of vascular dysfunction, is sufficiently uncommon to merit the recording of the following case.

Clinical Record.

The patient was a male, of Scottish ancestry, aged fifty-nine years, and a storekeeper by occupation. He was referred to me on June 5, 1950, because of right-sided abdominal pain, which he had experienced for the past fourteen years, and which had been less severe and persistent since his gall-bladder had been removed three years previously. In the course of the clinical inquiry he complained, although he was not unduly concerned, that the tips of the first three fingers of his right hand were painful when he rose in the morning, and also that they felt cold at this time; he had noticed these symptoms for some three months prior to examination, but he was indefinite as to the exact moment of their onset as they caused him no appreciable distress. He did not complain of any swelling, alteration in colour or disability when using the right hand, but rather he derived some relief from the pain when the hand was in use and after the fingers were warmed.

Some four weeks later, on July 4, I again examined this man at his special request, as he was distressed by severe and persistent pain in the above-mentioned fingers, which had also become cold, and further he noticed that he had much difficulty in picking up, and feeling, small coins on his shop counter with them; he said that the skin overlying the tips of the fingers concerned had become increasingly thickened and discoloured during the past fourteen days, and that he was no longer able to obtain relief by moving or massaging the fingers. When I more closely questioned the patient in regard to the condition of his left hand, he said that on occasions the fourth finger of this hand (the distal four-fifths) was subject to "painful spasms", in which he thought the covering skin was rather paler than normal; at the time of examination he was suffering no discomfort in this digit. Apart from the foregoing symptoms he had noticed that his memory and concentration were less acute than formerly, and his energy and enthusiasm were deteriorating; on occasions of mental and physical stress he complained of palpitations, but he was not unduly short of breath on effort, and he experienced no pain in the chest. For the past twelve months he had suffered a persistent cough with the expectoration of a moderate amount of sputum in the early morning; at no time had he coughed blood. He was able to perform his daily duties with comfort, and at no time did he experience pain in the lower limbs, at rest or on effort, by day or by night; his appetite was good and his weight constant at 14 stone 7 pounds; his skin was clear and at no time did he experience pain or swelling in the central or peripheral joints of the body. There were nc other significant symptoms referable to other systems. As a child he had suffered from "typhoid fever", but otherwise his past history and family health were uneventful; he had smoked some three ounces of tobacco per week for the past thirty to thirty-five years, and at no time had he been employed in a noxious trade; his alcohol intake was only light, and he denied venereal disease.

Physical examination of the patient revealed him to be a well-developed male of tall stature and good physique, who was not unduly concerned about his condition, but who was suffering obvious discomfort from the disability in his right hand.

There was no obvious deformity in size or shape of the right hand; the skin overlying the distal ends of the terminal phalanges of the first, second and third fingers was thickened and deep brown in colour, having the appearance of early dry gangrene; the largest area of tissue involved measured 1·1 centimetres in diameter, and this was present on the first finger; there was no swelling of the fingers of the hand. The skin overlying these fingers distal to a line through the mid-point of the middle phalanges of the first, second and third fingers was somewhat paler than the remainder of the hand, and palpation over this area revealed a pronounced alteration in temperature beyond this point due to the coldness of the fingers. There was some reluctance by the patient to flex the distal interphalangeal joints of the hand, otherwise movements were adequate and satisfactory. No abnormality in posture, size, skin colour or temperature was seen in the left hand. The walls of the radial and brachial vessels of both upper limbs were thickened and tortuous, but adequate pulsation was felt in both vessels, and this was regular in time and amplitude.

On examination of the lower limbs, full and forceful pulsation was present in the *dorsalis pedis*, posterior tibial and popliteal vessels of both lower limbs, and no undue thickening of their vessel walls was discernible clinically. The temperature of both lower limbs and feet was equal and normal, and there was no alteration in the colour of the skin when the limb was at rest, elevated, or held in the dependent position; no trophic changes were present. There was some increased tortuosity of the temporal vessels on both sides of the scalp.

The blood pressure was 160 millimetres of mercury, systolic, and 90 millimetres, diastolic; examination of the vessels of the *fundus oculi* revealed some evidence of early arteriosclerosis, with no venous engorgement; a well-developed *arcus senilis* was present in both eyes. The apex beat was palpable in the fifth left intercostal space, inside the mid-clavicular line; the heart sounds were audible in all areas, no abnormality of rhythm and no accompaniments being present. An occasional rhoncus was heard over both lung fields, otherwise no significant abnormalities were detected; there was a paramedian scar on the anterior abdominal wall.

Further clinical examination revealed no abnormalities in the other systems.

The following investigations were performed. Examination of the heart and lung fields by X rays and fluoroscopy revealed no abnormality. An electrocardiogram was within physiological limits. An X-ray examination of the upper and lower limbs revealed no evidence of calcification in the arterial walls, and no osteoporosis in the bones of the hands or feet was seen. Examination of the urine gave the following results: the specific gravity was 1015, the urine was acid and contained no albumin or glucose, and microscopic examination of the centrifuged deposit gave findings within normal limits. A full blood count revealed no abnormality in the number, size or shape of the red blood cells, and the hæmoglobin content was normal; the

white blood cells were normal in number and distribution; the blood failed to react to the Wassermann and Kline tests.

The diagnosis here was one of peripheral vascular lesion involving the vessels of both upper limbs, with accentuation of the disease process in the radicals of the right radial artery. As will be indicated later, it is almost invariable for an element of spasm to be added to the underlying vascular lesion, which, as in this case, leads to a final reduction in the blood supply, giving rise to the changes indicated and the early gangrene.

One hour after examination of the patient on July 4, 10 millilitres of 1% procaine solution were injected into the right stellate ganglion, and this procedure was followed by an immediate restoration of the normal colour and temperature in the affected parts and some 80% of his pain disappeared. A side-effect of the injection was a minor degree of brachial plexus involvement, and in addition a slight Horner's syndrome was evident; for the following two days he had a mild gastro-intestinal disturbance which he attributed to the procedure.

Some twelve days after this injection the patient declared that he no longer experienced any pain in the right hand or any subjective sensation of cold, and the range of movement of the fingers was satisfactory. There was no discernible alteration of temperature on palpation of the hand, and the skin over the ends of the affected fingers was healing and no longer the site of advancing gangrene.

When examined thirty days after the injection, the patient was well, and he declared that he suffered no discomfort or disability in his right hand. His psychological outlook was excellent. The skin over the five digits of the hand was normal in colour, temperature and texture, and the range of movement was not restricted.

Comment.

The purpose of reporting this case is largely to add emphasis to the occurrence of symptoms in the upper limbs due to *arteriosclerosis obliterans* in the absence of symptoms of vascular disease elsewhere. For this reason it is not intended to discuss any of the still controversial theories as to the ætiology and pathogenesis of this condition.

There is no known reason to suggest that the underlying vascular lesion has any selective cause when it affects different vessels; but rather it would be more acceptable to explain symptoms, when they arise, merely as the local expression of a widespread arteriosclerotic process.

In this case there is further evidence of the generally accepted opinion that in all cases of organic obliterative vascular disease the final factor causing complete obliteration of the already reduced lumen of the affected vessel is one of spasm, although it must be remembered that this element is essentially of secondary importance to the morbid process occurring in the vessel wall. The immediate relief of symptoms in this patient was really dramatic, as was the other evidence of an adequate circulation in the part. In this regard it is probable that such factors as exposure to cold and the use of tobacco were responsible for the initiation of this spasm; it is well known that in many cases of *arteriosclerosis obliterans* the capacity of the vessel to dilate is great, and when the stimuli initiating the spasm are removed the tonus of the vessel wall may return to normal for an indefinite period, which varies in different individuals. In regard to the long-term outlook for this patient, following the relief of symptoms by stellate ganglion block, one would hesitate to give a definite opinion or be moved to undue optimism by the initial gratifying response to sympathetic nerve block, as the management of these patients is still notoriously unsatisfactory. The local anæsthetic effect is usually only transitory, and permanent destruction of the ganglion by injection of a sclerosant, with its inevitable sequelæ, or surgical removal of the ganglion, would be the only hope of prolonged relief.

In this case the problem of future management is not so urgent as it would be if the lesion principally affected the lower limbs, as the part affected is more accessible and less vulnerable to trauma, and its use can be controlled.

As the condition of the affected part was satisfactory some five weeks after the injection of the local anæsthetic agent, treatment along the usually recommended conservative lines is advised, including the use of the vasodilator drugs "Priscol", 2-benzyl-imidazoline hydrochloride, and papaverine hydrochloride. With the almost inevitable development of further evidence of progress of the vascular lesion, on a clinical plane, further efforts to prolong an adequate circulation will have to be subsequently considered.

Acknowledgement.

I would express my thanks to Dr. Ivan Schalit, staff anæsthetist of the Royal Newcastle Hospital, who performed the block of the stellate ganglion in this case.

POLYRADICULONEURITIS IN A CHILD.

By D. C. HENCHMAN, M.B. (Sydney), D.C.H.,
London.

THE terms infectious polyneuritis, Guillain-Barré syndrome, radiculitis and infectious neuronitis are confusing, as well they may be until their ætiology and pathology are more definitely established. Some writers use these terms as synonyms, while others try to differentiate between them.

This subject has been excellently reviewed by Lewey (1945), who sums up the present position admirably. Briefly, the clinical condition he called infectious polyneuritis was described by Osler in 1892. In 1916, Guillain, Barré and Strohle published their observations on two cases of polyneuritis occurring in an epidemic amongst French troops. They considered that this was a particular type of polyneuritis and stressed the following features: (i) There was motor disturbance in the upper and lower limbs. (ii) The tendon reflexes were abolished, but the cutaneous reflexes were preserved. (iii) Parasthesia was present, but only slight disturbance of objective sensation, and pain on squeezing the muscles. (iv) The cerebrospinal fluid contained an increased amount of albumin without accompanying pleocytosis. (v) Complete and rapid recovery occurred.

Groups of cases have been described by other writers, some stressing facial paralysis as an important diagnostic feature, others finding in their series a mortality rate as high as 26·6% (Bradford, Bashford and Wilson, 1918). Guillain (1936) considered that most of these cases did not fulfil the criteria for his syndrome; but in 1937, as chairman of a symposium on radiculoneuritis, he felt compelled to widen the boundaries of the Guillain-Barré syndrome to include further types observed by himself or his co-workers.

Whether it is called infectious neuronitis or the Guillain-Barré syndrome, few cases of the condition in childhood have been published

Casamajor and Alpert (1941), in a review of the literature, cited 38 cases in the age group under twelve years in the English and French literature, and added a further three cases of their own. It would appear that the condition is being more frequently recognized. From 1942 to 1948, reports of 28 further cases in this age group appeared in the English-speaking literature alone, the largest series being twelve cases (Hatoff). In 1949 alone, reports of six cases were presented by Continental observers. In the same year Petch (1949) published his views on the Guillain-Barré syndrome, but he did not deal specifically with the age group I am considering.

Clinical Record.

I.A., a girl, aged four years, was admitted to hospital on September 15, 1949, with a history that she had fallen down three stairs four and a half weeks previously. She did not appear to hurt herself then, but two weeks later she woke at night complaining of pain in her legs. One week before her admission to hospital she developed a limp on the left side and appeared to be weak in this leg. She had had a poor appetite for two weeks and was listless. There was no poliomyelitis in the district at the time and other members of the family were well.

On admission to hospital, she was seen to be a big child for her age, and intelligent but very nervous. Her temperature was normal, and her pulse rate ranged between 100 and 160 per minute. No abnormality could be detected in the cranial nerves. The upper limbs were a little hypotonic and the full range of movement was present. The tendon jerks in the upper limbs were present on both sides. Gross ataxia and intention tremor were present. The abdominal reflexes were absent, and there appeared to be weakness of the trunk muscles, as the child had difficulty in sitting up. The child cried when the lower limbs were handled, but no localized tenderness could be demonstrated. Both legs were hypotonic, but with care the full range of all movements could be elicited. Both knee jerks and ankle jerks were absent, and the plantar reflexes were flexor on both sides. Sensation was normal to painful stimuli and to tickling, but lack of cooperation made further testing of sensation impossible.

A tuberculin skin test (Mantoux test, one in 1000) gave a negative result. X-ray examination of the skull, chest, hips and knees revealed no abnormality. The blood sedimentation rate was six millimetres in one hour (Westergren); the leucocyte count was 14,400 per cubic millimetre, the differential count being normal, and the Shick test gave a negative result. On lumbar puncture the pressure of the cerebro-spinal fluid was 190 millimetres of fluid (Queckenstedt's test gave a positive result), and examination of the fluid revealed albuminocytic dissociation (see Table I).

A week after her admission to hospital the patient was considerably more ataxic and was unable to walk. A provisional diagnosis was made of mid-line cerebellar tumour, despite the absence of papilloedema, and she was transferred to a neurosurgical unit. On September 26 ventriculography was performed by Dr. Gleadhill. Stereoscopic X-ray films were taken and revealed an entirely normal anatomy of the ventricular system. By October 7 she could sit up voluntarily but was unable to stand, and still complained of pain in her legs. The ataxia of the arms was less, and she used the hands in play and to feed herself. The child moved her legs voluntarily, flexing and extending both knees and hips; but the range of movement was limited. The greatest loss of power appeared to be in the quadriceps on both sides; there was some loss of power in the calf muscles and the *tibialis anticus* group also. The hamstrings appeared to have full power, and were in some degree of spasm. The muscles of both lower limbs were hyperæsthetic. Over the next few weeks the patient's condition improved gradually and the lower limbs became less painful. By October 28 she was able to stand with assistance and could make all leg movements against moderate resistance. Physiotherapy was instituted, and by the end of November she was able to walk without support, taking short steps on a wide base. Knee and ankle jerks were still absent, but otherwise neurological examination gave normal results. She was discharged from hospital on December 3 for further convalescence.

When she was last examined on February 16, 1950, the knee and ankle jerks were still absent. She was able to walk and run without stumbling and seemed as strong and active as any child of her age.

Comment.

The onset was gradual, with symmetrical paralysis associated with aches and pains, affecting mainly the proximal musculature. The deep reflexes were absent, the pulse rate was consistently high, and the cerebro-spinal fluid on repeated examination had a high albumin content and a low cell count. The functional recovery has been complete.

It is submitted that the above case fulfils the criteria for diagnosis of the Guillain-Barré syndrome.

Acknowledgements.

I am indebted to Dr. D. M. Douglas for his helpful criticism and for his permission to publish this case.

References.

Bradford, J., Bashford, E., and Wilson, J. (1918), "Acute Infective Polyneuritis", *The Quarterly Journal of Medicine*, Volume XII, page 88.

Casamajor, L., and Alpert, G. R. (1941), "Guillain-Barré Syndrome in Children; Review of Literature and Report of Three Additional Cases", *American Journal of Diseases of Children*, Volume LXI, page 99.

Guillain, G. (1936), "Radiculoneuritis with Acellular Hyperalbuminosis of Cerebro-Spinal Fluid", *Archives of Neurology and Psychiatry*, Volume XXXVI, page 975.

———, quoted by Lewey, F. H., *loco citato*.

———, Barré, J., and Strohle, A. (1916), "*Sur un syndrome de radiculo-néorite avec hyperalbuminose du liquide céphalo-rachidien sans réaction cellulaire. Remarques sur les caractères cliniques et graphiques des réflexes pendineux*", *Bulletins et mémoires de la Société médicale des hôpitaux de Paris*, Volume XL, Third Series, page 1462.

Hatoff, A. (1944), "Polyneuritis of Unknown Etiology in Childhood", *Journal of Pediatrics*, Volume XXIV, page 393.

Lewey, F. H. (1945), "What is Guillain-Barré Syndrome? Study of Underlying Pathological Lesions", *Journal of Pediatrics*, Volume XXVI, page 165.

Osler, W. (1892), "Principles and Practice of Medicine".

Petch, C. P. (1949), "The Guillain-Barré Syndrome or Acute Infective Polyneuritis", *The Lancet*, Volume II, page 405.

TABLE I.

Findings in the Cerebro-Spinal Fluid.

Date.	Pressure. (Millimetres of Mercury.)	Cells per Cubic Millimetre.	Protein Content. (Milligrammes *per Centum.*)	Pandy Test Result.	Sugar Content. (Milligrammes *per Centum.*)	Chloride Content. (Milligrammes *per Centum.*)	Cultural Examination.	Wassermann Test Result.	Lange Gold Curve.
16. 9.49	190	2	90	—	58	739	Fluid sterile	—	—
22. 9.49	150	2	120	—	73	688	—	—	—
26. 9.49	—	4	110	—	70	—	—	Negative	011211000
11.10.49	—	5	120	—	—	690	—	—	—
18.10.49	150	3	165	Positive	—	—	—	—	—
1.11.49	—	4	150	Positive	—	761	—	Negative	1112100000
30.11.49	110	2	45	Negative	—	690	—	—	—

DEATH FROM RESPIRATORY OBSTRUCTION IN HÆMOPHILIA.

By A. R. EDMONDS,
Medical Superintendent, Princess Margaret Hospital for Children, Perth.

THE following case report is of interest, particularly regarding the mode of death. There are a few reports of deaths from respiratory obstruction in hæmophilia, one by Boyd (1950), in which he refers to other reports of this occurrence. All these deaths occurred from spontaneous submucosal pharyngeal and laryngeal hæmatomata.

FIGURE I.

Clinical Record.

P.W., aged seven years, was admitted to hospital on October 16, 1949, at 8 a.m., with a history that two days earlier he had been knocked in the throat by the bar of a gate. Since then the neck had swollen and extensive bruising was observed. On the afternoon before his admission to hospital he had begun vomiting, and he had vomited frequently since. He had had nothing to eat or drink for eighteen hours, and could no longer swallow. He had difficulty in speech and some difficulty in breathing. No family history of abnormal bleeding was obtained at any time.

The child had first appeared at the hospital in November, 1942, with history of bruising easily and bleeding freely from small wounds. A blood count made at that time had given the following information: the erythrocytes numbered 4,600,000 per cubic millimetre; the leucocytes numbered 10,800 per cubic millimetre, 30% being neutrophile cells, 62% lymphocytes, 6% monocytes and 2% eosinophile cells; the platelets numbered 200,000 per cubic millimetre. The bleeding time was found to be two and one-quarter minutes and the clotting time two and one-half minutes.

Five other admissions to hospital were recorded between 1942 and the time of the patient's death, as follows. In September, 1945, he was admitted to hospital with hæmarthrosis of the right ankle joint and some bruises, mainly round about the legs. In November, 1946, he was admitted to hospital with a lacerated finger from which there was considerable hæmorrhage. This was stopped by pressure and elevation. A fortnight later, on November 22, 1946, he was readmitted to hospital with hæmorrhage from the same finger. In March, 1947, he had a few days in hospital following a fall from the back of a truck with some fairly

FIGURE II.

severe bruising. In November, 1948, he came to hospital with a large bruise on the hip, and since this appeared to be increasing, he was given a blood transfusion. He appeared a week later (November 22, 1948) with hæmorrhage from the wound in the right ankle where the blood transfusion had been given. This responded to conservative treatment. In between these times he was attended in the out-patient department on a few occasions. The blood investigations failed to reveal any ætiology for this hæmorrhage. The bleeding and clotting time estimation in September, 1948, showed the former to be four minutes and the latter three and one-half minutes. A fragility test made in November, 1946, revealed no increase; the result was about normal.

On examination of the patient on the occasion of his final admission to hospital, he was seen to have a grossly tense neck with obvious extravasation of blood into the skin, and he looked very ill indeed. It appeared as if he might die of asphyxia if the hæmorrhage was allowed to continue. It was decided to evacuate the clot (which was thought at the time to be mainly subcutaneous), in the realization that a tracheotomy might have to be performed at the same time. It was fully realized that he might have a laryngeal spasm, and preparations to cope with it having been made, anæsthesia was induced with "Pentothal". He stopped breathing during the induction,

and with great difficulty a gum elastic catheter was passed and breathing recommenced. The difficulty was occasioned by the inability of the anæsthetist to see the glottis. Eventually the tube was passed by digital rather than by visual examination. Tracheotomy was performed, the extravasation being observed to be deep and very extensive at the time of operation. He died the following morning, about fifteen hours after operation.

The only other finding of note, not recorded above, was the high specific gravity of the urine, which was 1·035; the urine was acid, amber in colour, and contained a large amount of sugar and a considerable amount of acetone.

Post-Mortem Examination.

Post-mortem examination revealed the patient to be a well-built little boy with multiple bruises on the limbs, a recent tracheotomy wound and gross extravasation of blood from the sternal notch to the chin. In the neck, extravasated blood penetrated all the layers of the cervical contents. The tracheotomy wound was clear and the trachea empty. There was a gross extravasation of blood over the epiglottis and vocal cords. In the chest there was an extravasation of blood throughout the mediastinum, which was most noticeable in the thymus and towards the roots of the lungs (see Figures I and II). Figure I shows the anterior view of the mediastinal structures and the viscera of the neck, which are completely black with extravasated blood. One can also see the hæmorrhages extending into the roots of the lungs on both sides. Figure II shows the posterior view of the tongue, epiglottis and opened œsophagus. The black appearance of the epiglottis and glottis, as viewed through a laryngoscope, made the visual location of landmarks in an emergency impossible.

Comment.

This child presented a typical clinical picture of hæmophilia; but at no stage did the bleeding and clotting times show the usual features of this disease, neither was there any family history. The mode of death is interesting, and it was unfortunate that further biochemical investigation were not carried out before death on the evidence presented by the high glycosuria. It is evident that some severe damage had occurred to the carbohydrate metabolism. It is possible that pressure on the thyreoid gland produced an overwhelming discharge of thyroxine into the blood-stream, depleting the liver of its glycogen stores. The urine test which was recorded was carried out shortly after the child's admission to hospital (which was on a Sunday), when his condition was reasonably good, and probably before the thyreoid gland had been put out of action by the hæmorrhage.

Death was ascribed to the mediastinal hæmorrhage. However, how this caused death is not quite clear. Contributing factors would seem to be: (i) pressure on cardiac nerves; (ii) pressure on phrenic nerves; (iii) interference with carbohydrate metabolism by hyperactivity of the thyreoid gland at first, and general depression of metabolism by the eventual probable destruction of that gland.

Bibliography.

Boyd, W. H. F. (1950), "Respiratory Obstruction in Hæmophilia; Recovery after Intubation", *The Lancet*, Volume 1, page 302.

REPORT OF TWO CASES OF TENOVAGINITIS STENOSANS.

By A. R. HAMILTON,

Sydney.

THE following two cases of *tenovaginitis stenosans* occurring in the right thumb in two sisters are considered worthy of record.

Case I.

J.N., a female patient, aged six years, was examined in 1944, when she complained of inability to straighten her right thumb. Her mother said that the condition had first been noticed when the child was aged two years, but would not say that full extension had been possible prior to that.

On examination of the hand a nodule was detected in the long flexor tendon of the thumb at the level of the metacarpo-phalangeal joint. Forced passive movement caused the thumb to extend, extension being accompanied by a loud click.

At operation the flexor sheath was incised longitudinally, whereupon the thumb extended readily. The *flexor pollicis longus* tendon, instead of consisting of a single tendon, was composed of three separate tendons in a single sheath.

A complete recovery resulted from the operation.

Case II.

R.N., a female patient, aged nine years, and a sister of J.N., was examined in 1950, when she complained of inability to straighten her right thumb. Her mother said that the condition had first been noticed when the child was aged three years, but would not say that full extension had been possible prior to that date.

On examination of the hand a tender nodule was detected in the long flexor tendon of the thumb at the level of the metacarpo-phalangeal joint. The thenar eminence was somewhat swollen, the appearance suggesting effusion in the sheath of the flexor tendon. When the proximal phalanx was fully extended passively, the distal phalanx was found to have a 90° flexion deformity, and when the distal phalanx was extended the metacarpo-phalangeal joint assumed a flexion deformity of 90°.

At operation the thickened flexor sheath was incised longitudinally just distal to the nodule, and the tendon, which consisted of a single bundle in this case, was found to be constricted at the level of the metacarpo-phalangeal joint, and immediately proximal to the constriction the tendon was considerably heaped up into a nodule.

Practically full extension resulted from the operation, a few degrees of residual deformity being due to uneven development of the articular surfaces at the interphalangeal joint.

Comment.

Most likely both cases were congenital in origin. The occurrence of this condition in two sisters, with the variations noted above, is of interest.

RUPTURE OF THE UTERUS IN THE THIRD STAGE OF LABOUR.

By R. FRANCIS MATTERS,

Honorary Gynæcologist, Royal Adelaide Hospital; Honorary Gynæcologist in Charge of Sterility Clinic; Honorary Obstetrician, Queen Victoria Hospital, Adelaide.

AT about midnight a medical practitioner rang me at my home and said that he thought that he had ruptured the uterus of a patient from whom he was endeavouring manually to remove the placenta. The doctor went on to say that he had sent the patient to my ward at the Royal Adelaide Hospital.

On admission to hospital the patient, pregnant for the second time and aged twenty-two years, was suffering severely from shock and her pulse was imperceptible. Her skin was cold and clammy and there were signs of air hunger.

The various resuscitative methods were instituted, including blood transfusion; but it was 7 a.m. before the patient was well enough to stand operation. It is difficult to decide the opportune time for operating in such a case;

but giving blood by transfusion to a patient who is losing blood rapidly is like trying to fill a leaking bucket—one must try to decide when the bucket is almost full and thus arrive at the opportune time. The transfusion was kept going during operation, which began at about 7.30 a.m.

When the abdomen was opened the large uterus, about the size of a six months' pregnancy, was delivered; it still contained the placenta. Posteriorly a jagged hole about four inches in diameter was found, and after aspiration of the blood from the peritoneal cavity, some fœtid-smelling gangrenous bowel was discovered; the smell was similar to that of an abdomen opened at post-mortem examination. Conservative treatment of the uterus was impossible, owing to the extensive trauma; also the anæsthetist continually repeated that the patient's condition was very bad. A total hysterectomy was performed and the hole in the upper part of the vagina repaired, enough of the vaginal vault being left open for drainage. Next the pelvic colon was mobilized. This was fairly easy, as the mesentery and *appendices epiploicæ* had been stripped off. What appeared to be viable gut was joined as proximal and distal parts of a colostomy spur. After a stab wound had been made in the left iliac region, the gangrenous loop was drawn through and the main abdominal incision closed. The loop of bowel was next clamped near the abdominal wall and divided by electric cautery. Soon afterwards the proximal loop was released and later the distal clamp was removed. A blood transfusion had been going all the time, and after the operation penicillin was run in, beginning with 500,000 units.

The patient gradually recovered. Later the loops were anastomosed by a general surgeon, and so the artificial anus was eliminated.

Comment.

The cause of the disaster was probably a failure to recognize where the uterine cavity was, and the lower uterine segment was thought to be a closed cervix. The hand was forced through the upper part of the vagina and the lower part of the thinned-out lower segment of the uterus into the peritoneal cavity. When the hand was passed into the peritoneal cavity, the practitioner, thinking that his hand was in the uterus, tried to scrape off the placenta; but what had really happened was that the *appendices epiploicæ* were scratched off the pelvic colon; thus hæmorrhage was caused and the blood supply removed, with the result that the bowel became black and gangrenous.

In a patient of this age one would have tried to save the uterus; but in the presence of the gangrenous gut and the jagged rent in the uterus the risk was too great. It is doubtful if this patient would have survived had it not been for the resuscitation and the chemotherapy.

Incidentally, the baby, it is understood, progressed without any setback.

Reviews.

THE MEDICAL ANNUAL, 1950.

ONCE again with the arrival of "The Medical Annual, 1950" we are able to urge our readers who have not done so to acquire the "Medical Annual habit".[1] This book, now in its sixty-eighth year of publication, is unequalled as a balanced and comprehensive review of the main field of medicine and surgery, especially for the general practitioner; in the present edition the editors, Sir Henry Tidy and Dr. A. Rendle Short, have maintained the standard. As well as the reviews of current literature on particular subjects by appropriate authorities, special articles and new material are featured. An important new arrival is the section on aviation medicine, in which is given a succinct account of the whole subject. An article on the Pulheems system of medical classification will supply most of the details required by those interested. A comprehensive classified bibliography provides guidance for the student of gerontology. Sir Philip Manson-Bahr surveys current views on the treatment of malaria and of leprosy and the use of antibiotics in the treatment of tropical disease. A series of interesting modern developments in veterinary medicine are considered in their relation to human medicine. Subjects of other major reviews include coccidioidomycosis, polyposis of the colon, medical and surgical aspects of gastric and duodenal ulcers, leucotomy, liver disease, occupational health, poliomyelitis, radiotherapy, chronic rheumatic diseases, social medicine, carcinoma of the uterus, and vesico-vaginal and recto-vaginal fistulæ. Lists of current additions to the ranks of pharmaceutical preparations and medical and surgical appliances, as well as new books, are useful features. The medical and surgical authors survey their respective fields in a brief but informative introduction, which is an important part of the book. "The Medical Annual" can more than justify its place on any medical practitioner's bookshelf.

[1] "The Medical Annual: A Year Book of Treatment and Practitioner's Index", edited by Sir Henry Tidy, K.B.E., M.A., M.D. (Oxon.), F.R.C.P., and A. Rendle Short, M.D., B.S., B.Sc., F.R.C.S.; 1950. Bristol: John Wright and Sons, Limited. London: Simpkin Marshall, Limited. 8½" × 5½", pp. 548, with illustrations, some of them coloured.

A TEXT-BOOK OF GYNÆCOLOGY.

THE third edition of a "Textbook of Gynæcology" by J. H. Peel follows the general plan of the previous edition and only minor alterations have been made in the text.[1]

Several sections have been revised and brought up to date. In the field of endocrinology certain methods of treatment which have not proved themselves have been discarded. The sections on endocrinology are well written and give a clear and concise guide of the source, preparation and clinical application of the hormones in gynæcology. The opinions expressed by the author are conservative and conform to the teachings held by the British school of gynæcologists. There is an excellent appendix of the endocrine products, and this should be a help to those that are confused with the various products marketed under different trade names by the pharmaceutical firms in this and other countries.

The general layout of this book is excellent, the diagrams and photomicrographs are of a high standard, and the index is comprehensive.

The practical details of treatment and general management are allotted more space than is usually given in a textbook of this size, and there is an excellent section on pre-operative and post-operative management with the treatment of the commoner post-operative complications. The operative section gives an abridged version of the techniques of various operative procedures, but this section is well illustrated and is adequate from the point of view of students. This section would require to be supplemented for those called upon to perform operative gynæcology.

This book is intended for the use of students presenting themselves for qualifying examinations and also for the general practitioner, and it can be recommended to them for its clarity of presentation and as an up-to-date, concise, orthodox book of British gynæcological teaching. There is no doubt that for students this is an excellent text-book.

[1] "Textbook of Gynæcology", by J. H. Peel, M.A., B.M., B.Ch. (Oxon.), F.R.C.S., F.R.C.O.G.; Third Edition; 1950. London: William Heinemann (Medical Books), Limited. 8¾" × 5¾", pp. 494, with 219 illustrations. Price: 24s.

A YEAR BOOK OF OBSTETRICS AND GYNÆCOLOGY.

FOLLOWING the general plan of the 1950 members of the Practical Medicine Series of Year Books, the editor of "The 1950 Year Book of Obstetrics and Gynechology",[2] J. P. Greenhill, opens the volume with a review of progress in obstetrics in gynæcology; he covers the period from 1940 to 1950, pointing out that the progress from 1900 to 1940 was discussed in the 1940 Year Book. Presented in this fashion the progress of the last decade is seen to be wide in scope and degree. Some matters are now so widely known and accepted that it is surprising to be reminded that they are of such recent origin; examples are knowledge of fœtal erythroblastosis and the Rh factor, and the effect of

[2] "The 1950 Year Book of Obstetrics and Gynecology (August, 1949-July, 1950)", edited by J. P. Greenhill, B.S., M.D., F.R.A.C.S.; 1950. Chicago: The Year Book Publishers, Incorporated. 7¾" × 5", pp. 580, with many illustrations. Price: $5.00.

maternal rubella on the fœtus; the use of antibiotics; the greater and safer use of Cæsarean section, especially the transperitoneal operation; advances in the early detection of uterine cancer, notably by cytological methods; new concepts and treatment of endometritis and progress in the investigation and management of sterility. The current articles abstracted, which are from journals received by the editor between August, 1949, and July, 1950, are arranged as in the previous Year Book: the section on obstetrics has chapters on pregnancy (physiology, abortion, ectopic pregnancy, complications and toxæmia), labour (general, anæsthesia and analgesia, complications, operative obstetrics and uterine hæmorrhage), the puerperium and the newborn; the section on gynæcology has chapters on general principles, diagnosis, infertility, operative technique, infections, benign tumours, special ovarian tumours, malignant tumours, menstrual disorders and endocrinology. Articles from a wide range of journals are abstracted, including six by Australian authors—F. Bellingham, R. Mackey and C. Winston, Eva A. Shipton, W. D. Cunningham, Charles Swan, Alan Grant, and A. A. Moon. The editorial comments are constructive and informative. The volume, which is admirably produced, will be useful to all practitioners concerned with obstetrics and gynæcology.

POST-MORTEM ROOM TECHNIQUE.

"PRACTICAL POST-MORTEM TECHNIQUE" by Edwin G. Poynter is a little book that helps to meet a definite need—instruction for the student post-mortem technician.[1]

The author, who has had an extensive experience at the Farnborough Hospital, Kent, describes in this volume the duties and work of the technician in the post-mortem room. He has set out to indicate normal post-mortem procedure, giving details of additional technique wherever necessary. He has succeeded in doing this clearly and concisely with the avoidance of technical language so that it can be easily followed by the student.

There is a simple introductory chapter on anatomy and physiology. A brief description of the instruments used and of their care is given and particular stress is laid on cleanliness in the post-mortem room. The author then proceeds to outline the technique for the removal of the various organs, mentioning the care necessary in medico-legal cases. There are useful chapters on the preservation of post-mortem material, the restoration of the body after accidents and the preservation of the body after death.

The book, which is suitably illustrated, reflects knowledge and experience; it is practical and will be found a useful guide for those who work in the post-mortem room.

MEDICAL SERVICE IN THE SUDAN.

THE title of C. E. G. Beveridge's book "Allah Laughed" is extracted from the Arabic saying "Allah made the Sudan and Allah laughed".[2] Apparently this saying intrigued Beveridge and was the final factor that took him into the Sudan Government Medical Service in 1925. A Victorian Rhodes Scholar, he had gone from the University of Melbourne in 1920 to New England College, Oxford, and Saint Bartholomew's Hospital, London. After 1925 he remained in the Sudan service until 1946, when he retired to live in Australia; he was successively Province Medical Inspector, Medical Officer of Health of several large provinces and Assistant Director of the Sudan Medical Service. "Allah Laughed" is the book his reminiscences, illustrated with his own photographs. Like most such books it is of uneven quality, though the assessment of the unevenness will vary with different readers. What is dull to some will interest others and *vice versa;* and incidents that seem rather pointless to many readers may well be rich memories to the author, who is as much entitled as the reader to enjoy himself in this particular type of book. However, the book is quite worth reading, especially if it is realized that the opening chapters are the least interesting—perhaps the story they tell is the furthest away in the author's memory and so has lost something of its freshness and detail. The closing chapters, notably the one on Sudanese staff and servants, are among the best.

Not the least of the book's virtues is its direct unpretentious style. The author allows himself a few clichés, but they are not objectionable, as they would be in a more ambitious setting. Only very rarely does he indulge in a platitude. Most of the book is a simple account of the life of the author and his wife in the Sudan and especially their tours around the country, complete with many anecdotes. Beveridge has a restrained but very real sense of humour and a humane outlook not only towards the people among whom he worked but also towards the wild beasts, of which he met many in his travels. He has not perhaps made as much of some of the more exciting incidents as would a less conservative writer prepared to use a few journalistic tricks. A map at the end of the book is liable to be overlooked and could with advantage be placed nearer the beginning. There are a few typographical errors, but in general the printing and production of the book are adequate. The photographs are interesting. The small black and white decorations at the ends of chapters are an attractive feature. This is pleasant holiday reading with a medical flavour.

KA-CHUNK, KA-CHUNK; OR, CARDIOLOGY FOR THE LAITY.

"ON many occasions", writes Dr. H. M. Marvin, in a preface to "You and Your Heart",[1] "after spending one or several hours gladly answering questions and trying to make clear to a patient the reasons for certain changes in his business or social activities, it has seemed to me essential that someone *should* write a book for those lay people. . . ." Thus, on learning from the publishers that such a book was projected and being invited to edit it, selecting such collaborators as might seem desirable, he readily accepted the invitation. The services of Mr. David Loth were secured to convert "the precious ore of difficult technical discussions" into "the shining metal of clear everyday words". The result is a handsome volume in which the intelligent layman may read as deeply as he pleases and in which he will learn that "the best advice for self-treatment that can be given is to follow as closely and as faithfully as possible the instructions of a competent physician". He will also learn that the heart as it beats produces "a sound something like 'Ka-chunk . . . Ka-chunk': the 'Ka' is the contraction of the ventricles, the 'chunk' the closing of the aorta's valve". The circulation of the blood, he will read was discovered by "an absent-minded British physician, William Harvey, who at one time was a tutor to the older sons of King Charles I (and was relieved of his post after letting the boys get too close to a battle while he was reading a book)". Other pieces of shining metal are that the heart in a lifetime exerts so much force that if it were all applied at once it could lift the biggest battleship afloat fourteen feet out of the water, and that women deposit their fat under the skin while men deposit it in the walls of the coronary arteries. By and large the exposition is a very good one, succinct, comprehensive and easily understandable. Gregg's discovery of the relationship between rubella and congenital cardiac defects is acknowledged. However, the subject of how atheroma begins is discussed without reference to important observations made within the last fifteen years, and the paragraphs about the relationship of effort to infarction of the heart are such as to discourage many who might successfully sue for worker's compensation. Opinions may differ as to whether it is a good thing to foster heart-consciousness amongst the laity. That the American Heart Association favours doing so is shown by the fact that this body exhibits display cards and bulletins in some 15,000 pharmacies conveying information about the heart and "carrying a 'see your doctor' message to approximately 3,000,000 people". The appearance of this book, edited by Dr. Marvin, who is President of the Association, is a

[1] "Practical Post-Mortem Technique: A Handbook for the Student Post-Mortem Technician", by Edwin G. Poynter, R.M.P., S.E.A.N., edited by Louis L. Griffiths, M.A., M.D., B.Ch., B.A.O., D.P.H., with a foreword by John F. Hackwood, M.D., F.R.C.S., F.R.C.S.(E.); 1950. London: Henry Kimpton. 6½" × 4¼", pp. 134, with 43 illustrations. Price: 9s.

[2] "Allah Laughed", by C. E. G. Beveridge; 1950. Melbourne: The National Press Proprietary, Limited. 7¼" × 5", pp. 268, with many illustrations.

[1] "You and Your Heart: A Clinic for Laymen on the Heart and Circulation", by H. M. Marvin, M.D., T. Duckett Jones, M.D., Irving H. Page, M.D., Irving S. Wright, M.D., and David D. Rutstein, M. D., with a foreword by Paul D. White, M.D.; 1950. New York: Random House. 8¼" × 5½", pp, 320, with a few illustrations. Price: $3.00.

further step in the same direction and indicates an expectation on the part of those qualified to know that many people will be found ready not only to accept but to purchase information about their hearts.

VENEREAL DISEASES.

IT is claimed that "A Text-Book of Venereal Diseases" by R. R. Willcox has a wide global outlook and should meet the requirements of venereologists in all climates.[1] The material is well arranged and the information up to date and adequate. There are numerous useful illustrations.

In covering his subject the author has had help from many sources and these are acknowledged in the preface. There is no bibliography.

Although only fifty-two pages have been allotted to gonococcal infections, all that is essential in regard to diagnosis and treatment has been included. The final chapter in the book, which deals with details of technique, contains, among other things, supplementary information concerning procedures in urethral irrigation, although in these days irrigations have a very limited use. Streptomycin is suggested instead of penicillin in cases of gonorrhœa with a probable concomitant early syphilitic infection, so that diagnosis may not be delayed by the possible masking effect of penicillin.

Non-gonococcal discharges in the male and female are discussed and Reiter's disease is noted very briefly.

The diagnosis and treatment of syphilis are considered in sufficient detail, and some good illustrations are included. Procaine penicillin is regarded as the penicillin of choice for treatment of syphilis, but in Great Britain no general reliance appears to have been placed on penicillin by itself, and in the services it is supplemented by arsenic and bismuth injections.

Arsenical and bismuth preparations are dealt with in special chapters and appropriate schedules of treatment are supplied. A point of interest is the mention that Indians "are very intolerant to the arsenicals, being prone to develop the often fatal complication of arsenical encephalopathy. Thus only penicillin and bismuth should be given to Indians".

There is an interesting chapter on "Yaws and other diseases resembling Syphilis", in which mention is made of Irkintja (Australian boomerang leg), which is a disease found in Australian aborigines.

The author's claim that the book has a wide global outlook is justified, for it may be recommended as useful anywhere.

A TRILINGUAL MEDICAL DICTIONARY.

THE late Dr. Samuel Johnson may have been perfectly correct in his statement that "to make dictionaries is dull work"; but to the reader who is interested in words, a dictionary can be a most fascinating book, apart altogether from its role as a tool of trade. Medicine, like all other arts and sciences, has its specialized vocabulary, growing and sprouting vigorously (and, be it observed, possibly needing expert pruning). People who deal with specialized subjects usually need to refer to literature in at least one other language than their own, and some like to use several foreign tongues. We have received a rather unusual type of medical dictionary in three languages—English, French and German.[2] It is divided into three sections—English-French-German, French-German-English and German-English-French, each arranged in strict alphabetical order in the first column. The dictionary is attractively produced, opens well and is easily consulted. The pages are printed in three columns, each in one of the three languages, with suitably differing type. For example, in the English-French-German section, the English column is printed in black type, the French column in light type and the German column in medium heavy type. This arrangement makes for clarity, and for ease in reading. The dictionary is concerned with names only; the terms are not defined. In the introduction the publishers give details of the methods that have been used in the compilation of the dictionary, and points to be observed in consulting it. Most of these require no comment, but one or two may be mentioned. For example, English compound nouns such as "brimstone liver", "hobnail liver" and so forth are listed as "liver, brimstone-", "liver, hobnail-", it being explained that the hyphen should be omitted when the expression is written. In the English section, "because of the enormous growth of American medical literature, the simplified American orthography has been used". In the English and French sections, normal anatomical terms in the German column are designated by the J.N.A. names; the B.N.A. names are given only in the German section of the dictionary. The names used by the British Anatomical Society not having been generally accepted, the terms hitherto used in British anatomy have been given beside them.

Switzerland, in which the English, French and German languages are all used, was undoubtedly a suitable country in which to produce such a dictionary as this, and the publisher is to be congratulated on having seen and seized the opportunity. No doubt many authorities will find things to criticize; but the editor, Dr E. Veillon, is well aware of his dictionary's limitations, and asks for suggestions from readers to help him and his team of collaborators to improve subsequent editions. They, and the publisher, have succeeded in producing a most useful work.

[1] "A Text-Book of Venereal Diseases", by R. R. Willcox; 1950. London: William Heinemann (Medical Books), Limited. 8½" × 5½", pp. 440, with many illustrations, some of them coloured. Price: 32s. 6d.

[2] "Medical Dictionary", by E. Veillon, M.D.; 1950. London: Lange, Maxwell and Springer, Limited. 9¼" × 6¼", pp. 1430. Price: £6 6s.

Notes on Books, Current Journals and New Appliances.

MEDICAL AND SOCIAL ASPECTS OF RHEUMATISM.

"THE SCOURGE OF RHEUMATISM" is a report of a conference held by the British Rheumatic Association in September, 1949, under the chairmanship of Lord Horder.[1] Each paper presented is followed by a report of the discussion. The opening paper is on modern views on the treatment of rheumatoid arthritis; it is brief but informative, and its author, W. S. C. Copeman, needs no commendation in the field of rheumatology. The remaining papers are mostly by lay people and deal with the care of the rural patient, the *Disabled Persons Employment Act*, "Remploy Limited" (a company formed to provide employment for disabled persons), the problems and employment of the housebound rheumatic sufferer, and the contribution of voluntary organizations. The background and much of the detail apply only to Great Britain, but the basic problem is the same the world over, and those who are concerned with it will find the views and experiences recorded stimulating and useful.

AN ANNUAL REVIEW OF MEDICINE.

FOLLOWING along the lines of the well-established "Annual Review of Physiology" and "Annual Review of Biochemistry", the first volume (for 1950) of the "Annual Review of Medicine" has appeared.[2] The place of this publication amongst the other books that cover current advances, and that appear either annually or as considered appropriate, is clear from the editorial preface. The purpose of the volumes, it is stated, is to review the various fields of medicine regularly, though not necessarily each year, unless the field and current advances warrant it. The policy is apparently to change the authors dealing with particular fields in succeeding volumes, with the object of avoiding biased interpretations. The reviews are intended "never to be expressionless listings containing little more than titles of papers". The authors have been asked to make their scope broad and to include their own critical assessment of work discussed, "to pitch their writings at a scholarly level, to include experimental background, and to avoid cookbook therapeutic recitals".

[1] "The Scourge of Rheumatism"; Report of a Conference held by the British Rheumatic Association, London, September, 1949; 1950. London: William Heinemann (Medical Books), Limited. 7" × 4¼", pp. 92. Price 5s.

[2] "Annual Review of Medicine", by Windsor C. Cutting and Henry W. Newman; Volume I; 1950. Stanford: Annual Reviews, Inc. 9" × 6", pp. 452. Price: $6.00.

The sections of the first volume deal with infectious diseases, diseases of the gastro-intestinal tract, the cardio-vascular system (medical and, separtely, surgical), the kidneys, the reproductive system, the nervous system (neurology), bones and joints (orthopædic surgery), the respiratory system, and the ear, nose and throat, nutrition in medicine, allergy, neoplastic diseases, obstetrics, psychiatry, physical agents and trauma, anæsthesia, radiology and radioactivity (radiobiology in the service of medicine), hæmatology (blood formation and the anæmias), laboratory aids to diagnosis and therapy, and therapeutics and toxicology. It will be obvious from this list that the term "medicine" in the title of the book is used in a broad sense and not as excluding surgery. The authors of the sections appear to have taken note of the editorial requirements already mentioned, and if this is maintained the annual volumes should obtain a ready welcome, supplementing and not supplanting those already accepted.

ANALGESIA FOR MIDWIVES.

E. H. SEWARD has written "Analgesia for Normal Childbirth" primarily for midwives registered under the *Midwives Acts*, 1902-1936.[1] It is based on the assumption that almost every confinement (in Great Britain) is attended by a midwife, acting either as a midwife or as a maternity nurse, and that she, with her intimate contact with the patient, must be relied on for the application of measures for the relief of pain in labour. The author discusses briefly the pain of labour and the methods and conduct of pain relief. Nitrous oxide-air analgesia is described in some detail with an account of the apparatus used and practical hints for the one who administers it. The last section states the rules framed by the Central Midwives Board with a direct relation to analgesia. The booklet is orthodox, brief and dogmatic, as is appropriate for its purpose.

PHYSICIAN'S HANDBOOK.

IN the sixth edition of "Physician's Handbook" the authors have maintained their standard in "summarizing briefly and clearly procedures and factual data that have proved most acceptable and useful to medical students and physicians".[2] The original material has been revised, expanded and rearranged in a more logical fashion. New sections have been added on electrocardiography, electroencephalography, radioisotopes, and simplified laboratory procedures. The purpose of the last-mentioned section is "to outline simple laboratory procedure that will extend and complement the physical examination of the patient"; it will be welcomed by those who, from necessity or choice, decide to carry out for themselves the simpler important laboratory investigations. The handbook is appreciably thicker than its predecessor, but still retains its convenient size despite the vast amount of material it contains. Reference to it is easier because of an improvement in printing and an ingenious rapid index system. It is an admirable little book that will be appreciated by students and practitioners of all grades of seniority.

THE CELL.

"CELL PHYSIOLOGY AND PHARMACOLOGY" by J. F. Danielli consists of six sections based upon lectures given at University College.[3] Danielli is well known for his earlier work on the nature of the cell membrane. The present book reflects his understanding of the cell in relationship to pharmacology, and is a very pleasant reflection of the maturity that he has attained in this field.

The first chapter on the cell as a physico-chemical unit provides a sound, but still imaginative, introduction to this field. From this beginning the author proceeds logically through the possible action of drugs on surfaces, the factors affecting the entry of drugs into the cell, and then the possible action of drugs on the enzyme systems of the cells. The fifth section deals with the possible manner of activity of narcotics, and finally the author deals with particular effects of drugs on cells.

Each section is well worked out, has a good bibliography and the necessary diagrams and illustrations for the purpose of the text. It is a book which will give a good introduction to general pharmacology to the beginner and occasionally tantalize the imagination of the experienced worker.

[1] "Analgesia for Normal Childbirth", by E. H. Seward, M.A., B.M., B.Ch. (Oxon.), D.A., D.Obst., R.C.O.G.; 1950. Oxford: Blackwell Scientific Publications. 8½" × 5½", pp. 30, with illustrations. Price: 2s.

[2] "Physician's Handbook", by Marcus A. Krupp, M.D., Norman J. Sweet, M.D., Ernest Jawetz, Ph.D., M.D., and Charles D. Armstrong, M.D.; Sixth Edition; 1950. California: University Medical Publishers. 7" × 4", pp. 388, with illustrations. Price: $2.50.

[3] "Cell Physiology and Pharmacology", by J. F. Danielli, Ph.D. (London and Cambridge), D.Sc.; 1950. New York, Amsterdam, London, Brussels: Elsevier Publishing Company, Incorporated. 9" × 6", pp. 166, with illustrations. Price: 24s.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"Recent Advances in Ophthalmology", by Sir Stewart Duke-Elder, K.C.V.O., M.A., D.Sc., LL.D. (St. And.), Ph.D. (London), M.D., F.R.C.S., Hon.D.Sc. (Northwestern), F.R.C.S. (Ed.), F.A.C.S., and Allen J. B. Goldsmith, M.B., B.S. (London), F.R.C.S.; Fourth Edition; 1951. London: J. and A. Churchill, Limited. 8" × 5½", pp. 388, with 139 illustrations, some of which are coloured. Price: 28s.

The last edition was published in 1934.

"A Synopsis of Surgical Anatomy", by Alexander Lee McGregor, M.Ch. (Edin.), F.R.C.S. (England), with a foreword by Sir Harold J. Stiles, K.B.E., F.R.C.S. (Edin.); Seventh Edition; 1950. Bristol: John Wright and Sons, Limited. London: Simpkin Marshall, Limited. 4¾" × 7¼", pp. 796, with 746 illustrations. Price: 25s.

The sixth edition was published in 1946.

"Diseases of Children's Eyes", by James Hamilton Doggart, M.A., M.D. (Cantab.), F.R.C.S. (England); Second Edition; 1950. London: Henry Kimpton. 9¼" × 6", pp. 326, with 211 illustrations, some of which are coloured. Price: 42s.

The first edition was published in 1947.

"A Synopsis of Anæsthesia", by J. Alfred Lee, M.R.C.S., L.R.C.P., M.M.S.A., D.A., F.F.A., R.C.S.; Second Edition; 1950. Bristol: John Wright and Sons, Limited. London: Simpkin Marshall, Limited. 4¾" × 7¼", pp. 368, with 66 illustrations. Price: 15s.

The first edition was published in 1947.

"The Care of Young Babies", by J. Gibbens, M.B. (Cambridge), M.R.C.P. (London), with a foreword by Sir Robert Hutchison, Bt.M.D. (Edin.), F.R.C.P. (London); Third Edition; 1950. London: J. and A. Churchill, Limited. 7" × 4¾", pp. 218, with 14 illustrations. Price: 5s.

The second edition of the book was reviewed in this journal on June 7, 1947; this, the third, edition has two new chapters on the underweight and overweight baby. The remainder of the text has been revised and partly rewritten.

"Juniors: A Book for Junior School Parent-Teacher Groups", by George H. Pumphrey, with a foreword by Sir Cyril Burt, D.Litt., D.Sc., LL.D.; 1950. Edinburgh: E. and S. Livingstone, Limited. 7½" × 5", pp. 186, with some illustrations. Price: 8s. 6d.

Deals with boys and girls between the age of seven and eleven years.

"Surgeons Twoe And A Barber: Being Some Account of the Life And Work of The Indian Medical Service (1600-1947)", by Donald McDonald, with a foreword by Lieutenant-General Sir Bennett Hance, K.C.I.E., O.B.E., K.H.S., I.M.S.; 1950. London: William Heinemann (Medical Books), Limited. 9½" × 7½", pp. 324, with many illustrations. Price: 42s.

The story of the Indian Medical Service in a popular form.

The Medical Journal of Australia

SATURDAY, FEBRUARY 10, 1951.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: surname of author, initials of author, year, full title of article, name of journal without abbreviation, volume, number of first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

THE VIRUS THEORY OF THE CAUSATION OF CANCER.

IT is not often that Australian medical practitioners have the advantage of hearing at their usual monthly meetings the opinions of those who have carried out important research on medical subjects in the Old Country. The presentation of the paper entitled "Recent Advances in Cancer Research" by Professor W. E. Gye and Professor Ida Mann at a meeting of the Western Australian Branch of the British Medical Association was thus an important occasion. The paper is published in the present issue. The views of Professor Gye and Professor Mann are most attractive, and it will be noted that, for them, the question of the cause of cancer seems to be determined. At the end of their paper they write that two lines of research are open. One is to find a substance which will kill the virus in its latent phase and so prevent the development of cancer even when secondary causes are present; the other is to find an antibiotic which will circulate in the blood-stream and kill the active virus particles, whether they are in the cells of the primary growth or in the metastases. Our present purpose is to bring to the attention of readers some recent work which has been published since Professor Gye and Professor Mann presented their paper in Perth.

It will be noted that Gye and Mann postulate two sets of causes. One group comprises the secondary causes and the other a primary or continuing cause. The secondary causes are the initiators or activators. They comprise carcinogenic agents ranging from heat, X rays and ultra-violet light through parasitic irritation to an increasing list of chemical substances including exogenous materials such as coal-tar derivatives and endogenous products of hormonal nature. The continued presence of these activators is not necessary for a continuance of the cell division. The primary or continuing cause, described by Gye and Mann as a unifying agent, is present in all tumours so far investigated, and in the opinion of Gye and Mann is a virus. Many speculations have been put forward in the past as to the nature of this continuing cause. Gye and Mann state that on the one hand it may be something inherent in the cell structure, something genetic, which predisposes it to react by continued division to a multitude of diverse stimuli. On the other hand, it may be exogenous, reaching the nucleus when its resistance is weakened by the secondary cause, and by continuing to multiply within the cell continually stimulate it to further mycosis long after the secondary cause has ceased to act. They state that the correctness of the second speculation is becoming increasingly obvious. They refer to four sign-posts which point towards their conclusion. It is not necessary to enumerate these, for they are clearly set out in the paper. It is on their experiment in the refrigeration and drying of tumour cells that Gye and Mann chiefly reply. In their present paper their views are described. Those who wish to investigate them further are referred to several other papers. One is by Professor Gye on the propagation of mouse tumours by means of dried tissue, published in the *British Medical Journal* of March 26, 1949, at page 510. The others by Professor Ida Mann comprise a group of three papers (W. J. Dunn is co-author of one) on the effect of low temperatures on the Bittner virus of mouse carcinoma, on the effect of repeated freezing and thawing on mouse carcinoma tissue and on the propagation of mouse carcinoma by dried tumour tissue, also published in the *British Medical Journal* on July 30, 1949. Our authors are hopeful that a substance may be discovered which has a specific action on the primary cause of malignant disease. They state that it is "lucky" that advance in our knowledge of cancer causation has come at a time when the finding of antiviral substances is a reality.

The recent work to which the attention of readers is directed has been published in the *British Medical Journal* of November 18, 1950; it has been carried out in the University of Leeds. The opening paper is by R. D. Passey and by L. Dmochowski. Since all members of the Branches of the British Medical Association in Australia receive copies of the *British Medical Journal*, it is to be hoped that they will study these three articles. To sum up the position shortly, it may be stated that Passey and his fellow author summarize the work of Gye and Mann and others in two conclusions: (*a*) that freezing alone is sufficient to kill tumour cells, and more effective still is freezing followed by drying; (*b*) that the newly induced tumour must have been initiated by a virus, since frozen and dried tumour material is able to induce tumours on inoculation. In their discussion they state that although from previous work it is clear that normal and tumour tissues can withstand freezing, even if embryo tissues cannot, yet in the face of the evidence Gye states: "The final conclusion we have reached from these studies is that malignant cells, like embryonic cells, are readily killed by extreme cold." In discussing the virus claim, Passey and his co-author point out that Gye *et alii* carried out no experiments to separate the supposed virus from the cellular element, nor did they test biologically for the presence of life in the tumour cells after freezing or freeze-drying. We can do no better than quote the summary of Passey and his co-authors:

First, in none of our centrifuge experiments were tumours induced with clear supernatant free from cells, while with few exceptions the sediments, in which apparently intact cells were always to be found, regularly induced tumours, which is clear evidence that the tumour-inducing activity was always associated with a tumour cell, and was not separable from it; secondly, the microscopic evidence provides further support for the belief that some of the cells had survived, in that, after the various treatments of freezing and freeze-drying, many intact cells were to be found which in appearance were very little different from the untreated minced material. It seems to us, in view of the available evidence, that there is no need to evoke a virus as the active agent.

The second paper is by R. D. Passey, L. Dmochowski and Ilse Lasnitzki and A. Millard. In their summary and conclusions they state that suspensions of sarcoma cells which have been frozen in glucose solutions and desiccated may show active growth *in vitro* after reconstitution in glucose. This, it is stated, indicates clearly that some tumour cells survive freezing and desiccation, and that the tumours which develop in mice injected with tumour material which has been frozen and desiccated, arise from such surviving cells. In the third paper, by Dmochowski and Millard, it is reported that suspensions of frozen and desiccated sarcoma cells embedded in clots formed by the addition of chick embryo extract to a mixture of rat and fowl plasmas were inoculated subcutaneously into mice. Sections of clots excised at varying intervals after grafting revealed the presence of viable tumour cells, increasing in numbers with the age of the graft. It should be noted that the articles from the University of Leeds are profusely illustrated by an interesting series of photomicrographs.

Work of this kind is as difficult as it is important, and it will be of interest to read any comments which Gye and Mann may make about the work from the University of Leeds. In an editorial in the *British Medical Journal* it is stated that it may be imagined that in this type of work almost any result might be obtainable according to the completeness of the desiccation. The editorial continues: "Despite these criticisms, most will agree with the authors' final conclusion 'that surviving cells and not a virus were responsible for the result attained by Gye and his colleagues'." The evidence points to the correctness of this conclusion.

Current Comment.

AMNIOTIC FLUID EMBOLISM.

AMNIOTIC FLUID EMBOLISM, two cases of which were recently reported from Melbourne by K. Bowden and E. Abrahams,[1] is rare, or at least rarely observed and recorded. It was first described in 1941 by P. E. Steiner and C. C. Lushbaugh[2] in a study of eight deaths associated with shock occurring during labour or shortly after delivery. Steiner and Lushbaugh listed as predisposing factors uterine tetany or exceptionally strong uterine contractions, meconium in the amniotic fluid, intrauterine death of the fœtus, an oversize baby, multiparity and advancing age of the mother. They found widespread embolism of small pulmonary arteries, arterioles and capillaries by the particulate matter in amniotic fluid and by meconium. The disease was duplicated clinically and pathologically in rabbits and dogs by the intravenous injection of human amniotic fluid and meconium. From the publishing of Steiner and Lushbaugh's observations to the time of writing a report of three cases of their own, G. K. Mallory, N. Blackburn, H. J. Sparling and D. A. Nickerson[1] had been able to find only 17 cases in the literature. All recorded cases (including their own three) had been diagnosed *post mortem*. It is interesting to note that at a meeting of the Victorian Branch of the British Medical Association held at Ballarat Base Hospital on November 25, 1950, E. R. G. Sheil presented a patient who had recovered after suffering from what all the evidence indicated was amniotic fluid embolism. Although the difficulties of clinical diagnosis are generally recognized, it is surprising that there have not been at least a few other reports of diagnosis before recovery or death. Steiner and Lushbaugh in their original report accepted the likelihood that diagnosis during life would become possible. Mallory *et alii* consider that "a gross diagnosis is difficult if not impossible, even for those familiar with the syndrome". However, at least a tentative diagnosis must be made if there is to be any hope of treatment.

What is to be the nature of rational treatment is not yet clear while many features of the disease remain obscure, but possibilities are opened up by a group of three studies from Boston[1] on the pathogenesis of the condition. In the first of these O. C. Leary and A. T. Hertig describe the results of examination of 14 placentas. In each case amniotic squamous cells, indicating the presence of amniotic fluid, were found within the placenta or its membranes; they lay between the amnion and chorion in nine cases, in marginal blood clot in two, in the decidua at the placental margin in one, in decidual sinusoids on the maternal surface in one, and in three of these sites in one. Leary and Hertig do not venture to guess which, if any, of these abnormal sites of amniotic squamous cells are important in the entry of amniotic fluid into the maternal circulation, but B. H. Landing, in the second of these studies, states that amniotic fluid appears to enter the maternal circulation only by way of uterine or placental vessels opened abnormally, as in *placenta accreta*, Cæsarean section, rupture of the uterus, partial retention of the placenta and, possibly, premature marginal separation of the placenta. The rarity of clinically evident amniotic fluid embolism, Landing points out, indicates that enough fluid to produce an embolic syndrome rarely enters the maternal circulation; but it seems possible that smaller amounts of amniotic fluid in the maternal circulation, or perhaps a slower rate of entry, can lead to a hæmorrhagic tendency. The relative contribution to the hæmorrhage of this factor (if it is significant) and of the primary condition (*placenta accreta et cetera*) cannot be deduced from the present evidence, but both may contribute. The point is that a generalized bleeding tendency can lead to death *post partum* despite adequate surgical treatment of local factors. This is elaborated by A. E. Weiner and D. E. Reid in the third of the studies. They found, from a review of 22 known cases of amniotic fluid embolism (published and unpublished), that of the 15 patients who survived delivery, 13 had hæmorrhagic manifestations before death. The suggestion is that amniotic fluid contains a factor responsible for the embolism syndrome and for the hæmorrhage. Weiner and Reid have shown that amniotic fluid contains an active coagulant factor, similar to thromboplastin in behaviour and entirely inactivated *in vitro* by small amounts of heparin. Other investigators have shown that the intravenous injection of thromboplastin into animals in large amount causes shock and rapid death, with intravascular coagulation in the larger vessels. In sublethal amounts it causes shock, but the animal survives with incoagulable blood; the blood has been defibrinated. Autopsy on these animals has revealed no thrombi in the large vessels, but extensive arteriolar and capillary thrombi, particularly in the pulmonary system. Weiner and Reid suggest that amniotic fluid normally plays a role in the coagulation of intrauterine blood and that this assists in post-partum hæmostasis. If it enters the maternal circulation, it may

[1] THE MEDICAL JOURNAL OF AUSTRALIA, December 23, 1950.

[2] *The Journal of the American Medical Association*, October 11 and 18, 1941.

[1] *The New England Journal of Medicine*, October 19, 1950.

cause intravascular clotting and consumption of fibrinogen, with post-partum hæmorrhage because of failure of the blood to clot normally. They consider that the gravity of the condition warrants consideration of any treatment "with a modicum of rationale"—no specific therapy is at present advocated—and tentatively suggest the use of heparin in a dosage of 50 to 75 milligrammes given intravenously. The hypothesis, based on the inactivating effect of heparin on the coagulant factor *in vitro*, is that this may decrease the clotting activity of the amniotic fluid in the maternal circulation. It is unlikely, they state, that this dosage would produce any alteration in the thrombin component of the clotting mechanism. The clinical indications for treatment would be cyanosis with sudden profound shock during labour or at delivery. Should the patient rally from the initial shock, hæmorrhagic manifestations may develop. On the basis that this may be due to afibrinogenæmia, venous blood should be examined for clot size and stability. The clotting mechanism should be restored by the giving of adequate amounts of fibrinogen (four to six grammes), with transfusions to replace blood lost. This is all tentative, but certainly warrants further investigation. Other possibilities will also need to be followed. Mallory *et alii* mention vascular spasm and anaphylaxis among suggested factors in pathogenesis; vasodilator or antihistamine drugs may be of value in treatment. Prophylactic ideas include the avoidance of violent labour in elderly *multiparæ*, especially after premature rupture of the membranes; "Pituitrin" needs to be administered with much discretion. Further investigation of local uterine and placental factors related to the entry of amniotic fluid into the maternal circulation may indicate surgical precautionary measures of value. Evidence has been put forward by C. L. Schneider[1] that, as a consequence of placental damage, material from the placenta, probably thromboplastin, may gain access to the maternal blood circulation and initiate clotting; this, too, may provide a profitable line of investigation. Amniotic fluid embolism is probably commoner than has been generally appreciated; its gravity warrants careful following of all trails likely to lead to its understanding and successful management.

DOSAGE FACTORS IN THE THERAPEUTIC EFFICACY OF PENICILLIN.

THE universal and everyday use of penicillin and the resultant familiarity with accepted standard dosage create a vague impression that all is known about dosage that needs to be known. However, H. Eagle, R. Fleischman and A. D. Musselman[2] have published results of laboratory investigation that should be heeded if penicillin dosage is to be considered rational. They refer to the apparently conflicting results obtained with various dosages administered at various intervals, results that have suggested, among other things, that the total duration of effective penicillin levels is not of critical importance; a high concentration acting for a short time has seemed to be just as effective as a low concentration acting over a longer period. From the experimental infections studied by Eagle, Fleischman and Musselman, it is seen that the curative dose of penicillin may vary to an extraordinary degree, in some instances more than a hundredfold, according to the method used for its administration. However, and this appears to be the important point, these "widely disparate but equi-effective curative doses" have in common the fact that in a given infection they provide the effective concentration of penicillin for essentially the same aggregate period of time, modified only by the varying effects of the penicillin-free interval between injections. For a significant period of time after the serum penicillin concentration has fallen to ineffective levels, the surviving but damaged organisms fail to multiply, and with some species they remain susceptible to the host defences. Eventually they recover and resume multiplication. The longer further treatment is delayed, the greater is the degree to which remultiplication during the penicillin-free interval counteracts the effect of the preceding treatment. The degree of delay possible without serious adverse effect varies with a number of factors. First, the danger is obviously greater with a rapidly multiplying organism than with one that multiplies slowly, for example, the organism of syphilis. Secondly, the concentration of penicillin to which the organisms have been exposed, and the duration of that exposure, affect the time required for the bacteria to recover. Thirdly, with bacterial species that recover from the toxic effects of penicillin only slowly, the interval can safely be much longer than with species that recover quickly. Finally, the smaller the number of organisms that survive the preceding treatment, the less harm is done by remultiplication for the first few generations. The fact that bacteria remain static in number or may continue to die for a considerable period after the penicillin concentration has fallen below the bactericidal level constitutes an important safety factor in the therapeutic use of penicillin. Nevertheless, a schedule of treatment that provides the maximally effective bactericidal concentration at the focus of infection continuously may well be expected to be more rapidly effective than a discontinuous schedule.

The point of this discussion is that, as Eagle, Fleischman and Musselman point out, adequate dosage with penicillin has at least two parameters. One is the concentration attained at the focus of infection; the other is the total time for which that concentration is provided. There is no particular virtue in concentrations of the drug in excess of the maximal bactericidal concentration; large doses are more effective than smaller primarily because they provide the effective concentration for a longer time. Penicillin suspended in absorption-delaying vehicles that provide a reasonably sustained but low level should be just as effective as aqueous solution given in multiple injections, provided only that the absolute concentration attained at the focus of infection is in excess of that necessary to kill the particular organism at the maximal rate. The possibility must be borne in mind that the concentration may drop below the level effective for a particular organism after a certain period of time. The practice of incorporating a small amount of sodium penicillin in such preparations would appear to add little to their therapeutic efficacy. With organisms for which the procaine penicillin salt provides adequate concentrations the short-lived high concentrations afforded by the sodium salt are unnecessary and do not accelerate the death of the organisms. With organisms not affected by the low concentration of the procaine salt, the sodium-procaine mixture will be effective only for the time that the sodium salt provides the high concentrations necessary for the particular organism; treatment must therefore be repeated as often as if the sodium salt was used alone. A special situation is offered by an area of chronic inflammation with a walled-off focus of infection only slowly equilibrated with the serum and the surrounding tissue fluids; here it may be necessary to keep the serum concentration at a relatively higher level for a longer time to ensure an effective concentration at the actual focus of infection. These ideas, with their experimental basis, are of considerable interest and should cause some fresh thinking about penicillin dosage. It is clear that overdosage, underdosage and unsuitable intervals between injections can all result in inefficient therapy and waste of penicillin.

[1] *Surgery, Gynecology and Obstetrics*, May, 1950.

[2] *The American Journal of Medicine*, September, 1950.

A SIGN OF THE TIMES.

THE head of a department in the University of Sydney who wished to return a book to the office of THE MEDICAL JOURNAL OF AUSTRALIA explained his delay by writing that he had no messenger boy—the boy "having accepted a job in a cosmetic factory at a senior lecturer's wages".

Abstracts from Medical Literature.

SURGERY.

Plastic Reconstruction of the Anterior Thoracic Wall.

D. A. CAMPBELL (*The Journal of Thoracic Surgery*, March, 1950) describes a method of repairing large defects of the anterior thoracic wall, which are left after the removal of memmary and other cancers invading the bony thorax. The method involves use of a graft from the *fascia lata*, the *latissimus dorsi* muscle (which is completely mobilized and transplanted) and split-thickness skin grafts.

Pharyngeal Diverticulum.

SIR THOMAS DUNHILL (*The British Journal of Surgery*, April, 1950) gives his experience in 28 cases of pharyngeal diverticulum; 21 of the patients had been operated on. The anatomy, ætiology, symptoms and diagnosis are discussed and the literature is summarized. The author now does a single-stage operation, using a collar incision and dissecting the pouch to its junction with the œsophagus. The two layers forming the diverticulum are separated just at the neck and sutured separately. The details of the technique are given and the operative procedures are illustrated. Other methods of operation are discussed, and advice is given on the management of such a case.

The Value of the Fibrinogen B Test.

E. R. TRETHEWIE, R. D. CARMAN AND A. J. DAY (*The British Journal of Surgery*, July, 1950) report investigations to determine the presence of fibrinogen B in patients in a surgical ward. In approximately 50% of cases the result of the test was found to be positive. Of the patients with a positive result, half were given a prophylactic course of heparin. Symptoms and signs of venous thrombosis and pulmonary infarction developed in members of both "fibrinogen B positive" and the "fibrinogen B negative" groups. No untoward symptoms developed in the patients who were given prophylactic heparin therapy after treatment was instituted, though some had developed symptoms or signs of thrombosis or infarction immediately before the administration of heparin. Clotting times of the blood of patients were estimated at 37° C. and at room temperature in many instances. No significant help as to the imminence of thrombosis was obtained from this investigation. The authors concluded that estimation of the fibrinogen B was not of any particular practical value. Their experience was that a smaller dose of heparin than that usually advised, especially with the regard to the frequency of dosage, was satisfactory in cases of embolism and thrombosis.

Blind Intestinal Pouches Resulting from Lateral Anastomosis.

CARL J. HEIFERZ AND H. R. SENTURIA (*Surgery*, May, 1950) describe eight cases of blind intestinal pouches following lateral anastomosis. They state that Cannon and Murphy in 1906 reached the following conclusions: (i) lateral anastomosis was unphysiological, since it severed the circular muscle fibres at the anastomotic site and interfered with the proper propulsion of the intestinal contents; (ii) the stasis thus created tended to enlarge the blind end of the intestine extending beyond the opening; (iii) to prevent this pouch the anastomotic opening should be carried as close to the inverted ends as possible; (iv) end-to-end anastomosis avoided this hazard. The authors give the case histories of eight patients, in five of whom the symptoms were considered undoubtedly due to the blind pouch. In the other three patients, other lesions were present which could have produced the symptoms. The symptomatology falls into the general categories of perforation with peritonitis, intestinal obstruction, nutritional disturbances and anæmia. They consider that careful X-ray studies should be carried out on all subjects of lateral anastomoses who suffer from symptoms such as abdominal pain, distension, nausea, vomiting, diarrhœa and nutritional disturbances. The need for surgery depends on the nature of the symptoms and technical considerations. The substitution of end-to-end for lateral anastomoses prevents the formation of blind pouches.

Phantom Limb Pain and Central Pain.

T. T. STONE (*Archives of Neurology and Psychiatry*, May, 1950) discusses the various types of pain and other abnormal symptoms associated with phantom limbs. He regards the symptoms as having an organic basis if they are undue warmth, itchiness or distress in the phantom limb, intermittent or continuous severe burning pain, deep agonizing, torturing sensations, piercing, cramping or cutting pains, and various throbbing or stabbing pains. These symptoms occurred more in older patients and more frequently in upper than lower limb amputations. For this type of symptom he suggests a subpial resection of the appropriate area in the posterior central convolution as most likely to relieve pain. His conclusions are based on experience with three patients.

Tumour Formation in Atrophic Testes.

J. S. HAINES AND HARRY GRABSTALD (*Archives of Surgery*, May, 1950) report two cases of testicular tumour, both associated with previously existing atrophy of the involved testis. They state that testicular cancer accounts for approximately 1% of all deaths from cancer in males. Little is known of its cause. One of the few known factors is the increased incidence of carcinoma in patients with pre-existing ectopy or cryptorchidism. In groups of adult men examined, the incidence of undescended testes was found to be approximately 1%; in boys under sixteen years of age it is said to be 4%. Other figures quoted are that 12% to 14% of all undescended testes will become involved by tumour, and the formation of tumour is said to be 150 to 200 times more common in the abnormally located testis than in scrotal testes. Even a more conservative figure records testicular tumours as 22 times more common in undescended testes. Since orchidopexy has not significantly lowered the incidence of tumours as shown by the work of Gilbert, it would seem that cryptorchidism *per se* is not the predominant ætiological factor in testicular tumours. The following factors are quoted: (i) in the general male population, the incidence of atrophy is as great as the incidence of undescended testes; (ii) testicular tumours are more common in undescended than in normally placed testes; (iii) undescended testes are almost always atrophic to some extent; (iv) testicular tumours are more common after non-specific atrophy of the testes than after mumps orchitis with atrophy.

Uræmic and Trophic Deaths Following Leucotomy: Neuroanatomical Findings.

TURNER MCLARDY (*The Journal of Neurology, Neurosurgery and Psychiatry*, May, 1950) analyses the causes of death following leucotomy. Death from cerebral hæmorrhage occurs within fourteen days after operation with an average of five days, and death from intracranial infection within sixteen days with an average of ten days. After these patients are excluded, there are two other groups. One is of those dying predominantly from uræmia in the third post-operative week; this group has bilateral damage to the posterior non-granular orbital cortex, posterior area 47. Another group die predominantly from trophic disorders with marasmus, skin bullæ and trophic skin lesions between two and five months after operation; these deaths were associated with the bilateral cutting of the region of the subcallosal fasciculus at the level of the head of the caudate nucleus.

Surgery in Psychoneurotic Women.

PEARL C. ULETT AND EDWIN F. GILDEA (*The Journal of the American Medical Association*, July 15, 1950) have compared the incidence of major surgical procedures in 214 case histories of chronic psychoneurotic women with the incidence in a group of 100 control subjects. Of the members of the psychoneurotic group 60% had undergone major procedures and 43% had undergone some major gynæcological operation. In the control group 32% had had major surgery and 15% had had a major gynæcological operation. The authors discuss the various factors which may help to explain the higher incidence of surgery in the psychoneurotic group and suggest the following possible means of decreasing the number of unnecessary surgical procedures in the group. Firstly, they emphasize the importance, especially for the neurotic patient, of not overemphasizing unimportant abnormalities. Secondly, they consider it important that emotional problems be recognized and that their more obvious relations to the patients' complaints should be explained. Thirdly, they state that except when symptoms are acute, surgery should be avoided in a psychoneurotic person until the emotional factors have been investigated. If organic disease does require surgical treatment, the patient should receive both pre-operative and post-operative psychotherapy.

Immediate Cholangeography.

R. F. CARTER AND L. GILLETTE (*The Journal of the American Medical Association*, July 15, 1950) describe a method of determining the exact configuration of anatomical lesions of the

biliary tract, which they consider simple, reliable and safe. They recommend the use of immediate cholangeography in congenital obstructions of the biliary duct, in those obstructions due to calculi or stricture, and whenever there are indications for exploration of the common bile duct. They describe cases illustrative of the usefulness of immediate cholangeography.

Chronic Backache in Compensation Cases.

SAMUEL KLEINBERG (*The American Journal of Surgery*, August, 1950) discusses the duty of the physician in relation to the patients with chronic backache who are a trial to the physician, the insurance carrier and the labour department. He lists the many causes of backache and discusses the method of examination in order to appreciate the many causes and the influence of trauma or preexisting pathological disease in the back. The examination must be thorough soon after the injury to establish a definite diagnosis. Accurate and adequate treatment should be instituted promptly. Exaggeration of symptoms by the patient should be discouraged, and the use of a frightening diagnosis should be avoided. The physician and the insurance carrier would do well to utilize early consultation with an orthopædic surgeon and if necessary with a neuropsychiatrist. Unduly prolonged treatment, especially physical therapy, should be avoided.

Carcinoma of the Sigmoid Colon with Prolonged Survival.

H. A. SOLOMON AND S. I. KREPS (*The Journal of the American Medical Association*, September 16, 1950) report a case in which the patient survived for twenty-six years and two months after the resection of an adenocarcinoma of the sigmoid colon. A slowly growing solitary liver metastasis was discovered at least three years before the death of the patient. Except for that metastasis and metastases in the perihepatic and periaortic lymph nodes post-mortem examination disclosed no other primary neoplasm or evidence of recurrence of the original tumour. The patient was never invalided by the long course of neoplastic disease. She died of massive hæmorrhage into the gastro-intestinal tract from the rupture of an œsophageal varix, which had developed as a result of portal obstruction produced by the pressure of the liver metastasis.

The Vagus Nerve in Cardiac Arrest.

H. E. SLOAN (*Surgery, Gynecology and Obstetrics*, September, 1950) states that in a series of experiments on 36 dogs, strong faradic stimulation of the intact vagus nerve at the hilum of the lung under conditions of adequate oxygenation failed to produce cardiac arrest. In 30 dogs subject to severe progressive hypoxia or asphyxia central cardioinhibitory impulses were present which resulted in increasing bradycardia and a fall in blood pressure. Stimulation of the vagus nerve under such conditions did produce additional inhibition of the heart, which was frequently followed by temporary cardiac arrest. This phenomenon occurred only in extreme degrees of oxygen deficiency. The author suggests that vagal reflexes arising during intrathoracic operations are of little importance in well oxygenated patients. They may, however, play a part in augmenting an existing oxygen deficiency and may aid in producing cardiac arrest under conditions of severe hypoxia or asphyxia.

Acute Cholecystitis.

P. ROSS, J. D. BOGGS AND J. E. DUNPHY (*Surgery, Gynecology and Obstetrics*, September, 1950) state that a review of material from the Peter Bent Brigham Hospital indicates that resolution of the inflammatory process after an attack of acute cholecystitis is extremely slow and incomplete, regardless of the clinical course of the patient's condition. After a short period devoted to restoration of fluid balance and the beginning of antibiotic therapy, there is nothing to gain by delaying surgery if the patient is doing well. The inflammatory process persists for months and sometimes for years after the clinical symptoms subside. The period spent in hospital is prolonged and the technical difficulties of surgery increased. On the other hand, if the patient is doing badly surgery is mandatory, and it is in such cases that simple cholecystectomy under local anæsthesia may be lifesaving. The authors consider that the concept should be abandoned that there is any arbitrary critical period during which surgery for acute cholecystitis is to be avoided.

A New Concept of Venous Thrombosis.

A. J. QUICK (*Surgery, Gynecology and Obstetrics*, September, 1950) offers a simple explanation for the formation and propagation of the thrombus within a vein. He states that as a result of an injury to the vessel wall, even though it is a minor one, a rough surface is produced to which platelets adhere. As these cells disintegrate thrombin is produced and the formation of a thrombus is initiated. This primary thrombus undergoes contraction and exudes a serum rich in nascent thrombin. If the circulation is sluggish this serum will not be washed away promptly, but instead it will cause the formation of the secondary thrombus upon the original fibrin clot. As the new addition to the primary coagulum retracts, it gives rise to further growth of the thrombus. Clot retraction not only explains the propagation of the clot, but it also accounts for its non-attachment to the vessel wall except at the site of origin. It can be postulated that any means which diminishes or slows clot retraction should be valuable prophylactically against thrombosis. The reduction of the number of circulating platelets is a theoretical means, but it is not feasible clinically. The reduction of the potential formation of thrombin is readily accomplished with heparin and with dicoumarol. Particularly important as a prophylactic measure is the correction of anæmia, since the lower the cell volume the greater the retractility of the clot.

Spontaneous Pneumothorax.

F. S. DOLLEY AND L. A. BREWER (*The Western Journal of Surgery, Obstetrics and Gynecology*, September, 1950) state that chronic spontaneous pneumothorax is a potentially serious condition, and that without treatment leading to lung reexpansion the effect may be very disabling. The condition is due with few exceptions to the rupture of a congenital cyst, pleural cyst, emphysematous bleb or bulla. The consideration with each case of the most probable ætiological factors is of therapeutic importance. Surgery is indicated for its relief. Maximum pulmonary function should be preserved because of the danger of a contralateral pneumothorax or of an extensive inflammatory process. If a ruptured congenital or emphysematous cyst is found, its wall should be excised. If a subpleural bleb is encountered, complete pulmonary lysis usually effects a cure in addition to the closure of the pulmonary opening responsible for the pneumothorax. In all cases all pulmonary adhesions, both parietal and, if possible, interlobar should be divided. The authors report the presence in 10 of their cases of an inelastic fibroblastic visceral pleural restraining membrane covering the lung. They state that without the removal of this structure return to satisfactory function of the collapsed lung is not possible, regardless of the excellence of pleuro-pulmonary repair.

Primary Carcinoma of the Liver.

KENNETH E. LEMMER (*Archives of Surgery*, October, 1950) reports a study of 18 cases of primary carcinoma of the liver. Fifteen of the subjects came to autopsy; three diagnoses were made by laparotomy. Of the 18 patients, 17 have died and one only is living five months after resection of a solitary hepatoma. The average duration of symptoms in the 17 fatal cases, from their onset to the death of the patient, was seven months. The cases comprised 11 hepatomata with cirrhosis of the liver in seven cases, six cholangiomata with cirrhosis in four, and one cholangiohepatoma with cirrhosis. The incidence shows that primary carcinoma of the liver is an uncommon condition of the white race, although the incidence is high in the Chinese and East African natives. It is a disease of older persons and the lesion is highly malignant. It is commonly associated with cirrhosis of the liver, and the usual signs and symptoms of intraabdominal carcinoma are present. In this series a clinical diagnosis of primary carcinoma of the liver was not made.

Hypospadias.

ARTHUR A. SCHAEFER AND JOHN ERBES (*The American Journal of Surgery*, August, 1950) review 62 cases of hypospadias in patients at Milwaukee Children's Hospital between 1924 and 1948. In 16 cases, operation was not thought necessary or permission to operate was not granted. Forty-six patients underwent operations. In this series 32% of cases were glandular in type, 62% penile and 6% perineal. In 10 cases a meatotomy only was carried out; in three cases the Nove-Josserand operation was performed, in three the Duplay and in 25 the Ombredanne. Five patients had had the first stage (chordee) only performed and awaited urethral reconstruction. Of the three patients treated by free skin tube grafts the result in one only was satisfactory, as it was in only one patient of the three treated by skin flaps (Duplay-Thiersch method). Of 25 cases in which the third stage of the Ombredanne operation had been completed the authors considered the result in 19 satisfactory.

British Medical Association News.

A MEETING of the New South Wales Branch of the British Medical Association was held at Broughton Hall Psychiatric Clinic, Leichhardt, New South Wales, on December 7, 1950. The meeting took the form of a series of clinical demonstrations by the medical superintendent, Dr. Guy Lawrance, and members of the staff of the clinic. Parts of this report appeared in the issues of December 23, 1950, and January 13 and 20, and February 3, 1951.

Puerperal Psychosis.

A patient was presented suffering from puerperal psychosis of a schizophrenic type. The patient, a married woman, aged twenty-five years, a *multipara*, had been admitted to the clinic on July 28, 1950, because she had become very strange in manner and had been detected in the act of drinking poison. Her first child had been stillborn, but the last baby had been delivered successfully five months previously, and the present illness dated from that time. She had become depressed, moody and morose and had developed severe insomnia. She said that her husband looked pale and listless and appeared to be watching her all the time. Two months before her admission to the clinic she had developed auditory hallucinations and heard voices urging her to take poison. She said that later God's voice instructed her to commit suicide by taking poison. That she attempted, but she was interrupted and taken to the Reception House. There the voices left her, but she expressed delusions of a somatic nature and believed that she had kidney disease, cancer, consumption and venereal disease. She had a course of electroconvulsive therapy and was sent home, but her condition soon relapsed. She became depressed and lachrymose and wandered aimlessly away from the house, and it was feared that she would take her life. On her admission to Broughton Hall she was crying and depressed, but she again had auditory hallucinations which told her that her husband was going to leave her. She expressed a delusion that her baby had been changed for another one, and she felt that she would kill it. She was retarded, and had ideas that people stared strangely at her. She had no interests, was slow to obey instructions and sat idly about gazing into distance. She gave a very poor story of her recent troubles, spoke in a dull, monotonous tone and was not very cooperative. It was considered that she had a severe schizophrenia reaction with strong depressive features. The puerperium was a precipitating factor, freeing the mental illness in a person predisposed to the condition. At the age of eighteen years the patient had been discharged from the Australian Women's Army Service because of neurosis. In her earlier life she had been happy until the age of thirteen years, when her parents died within a few months of each other, the father of cardiac disease and the mother of cancer. The four siblings were scattered to the care of different relatives. At the age of fourteen years she went to business college, and she had had several positions prior to her marriage and had given signs of having a stable personality. She read many books on sex, and gained enough ideas to satisfy herself and to instruct her husband in his part. After the death of her parents she had a hard life, and had to support herself until the age of fifteen years. She brooded a lot over the hardships of those early days. Her husband was a Roman Catholic, and told her that he regretted having left his faith for hers (she was a Methodist) and having allowed her to have the children christened in her religion. Two years before her admission to the clinic her younger brother had been killed in an accident, and altogether she had had enough to upset a stronger-minded person. The reaction was a severe one, and early treatment had not been persevered with for long enough. Electroconvulsive therapy was given, with rest and sedation. If the schizoid element did not clear up, full insulin coma therapy would be given. The prognosis was not favourable, because of the dissociation factors present and the unsatisfactory early career of harsh childhood experiences. The death of her first child and the death of her brother were important factors. Her physical health was fairly good. It was pointed out in conclusion that the childbearing period was not productive of any specific type of mental illness, but that the stresses and strains involved set free the illness which the particular patient was prone to develop.

Reactive Depression.

A single woman suffering from reactive depression was presented. She was aged fifty-six years and was a domestic by occupation. She had been admitted to the clinic during April, 1950, with a history of depression accompanied by a suicidal attempt. One of her brothers was in a mental hospital, a sister and brother had died of "stroke", and four siblings had died at early ages. Her mother had died when she was aged six years, and she was reared in an orphanage. At the age of seventeen years she was sent out to domestic service. She had had one love affair when she was aged twenty years, but it came to an unhappy end and she had made no other heterosexual attempt. She had kept her positions for long periods, her main relaxation being regular attendance at the cinema. She had a few female friends, and was fussy, over-tidy and easily irritated by trifles. She regretted having remained single and was jealous of her married sisters. She often talked about her boy friend of thirty-six years before. Her menopause had taken place seven years before her admission to the clinic, and about three years before her admission she had suffered from measles, from which she did not make a good recovery as her heart had not been satisfactory since. When she was told that her heart was affected and that she should not work any more, she dropped her initiative, and, refusing to ask for an invalid pension, she began to live on her savings. She became very depressed, gave up her friends, could not sleep and became unable to fend for herself. She also became restless, and said that she would never be better. She asked for poison, but climbed out of a window one night with the intention of drowning herself in the dirt tin, which was full of water. However, her courage failed her, and three weeks before her admission to the clinic she gave up the struggle and entered hospital. Since entering the clinic she had been a little retarded, but had been restless and dissatisfied. She expressed self-pity because of her loneliness and lack of friends to stand by her. Her condition was regarded as a depressive state occurring in a person whose whole life had been a drab and uninteresting experience. She had been suffering from lack of heterosexual love and had lacked the drive necessary to seek it. The menopause had no doubt unsettled her, and then a weak heart resulting from measles had given her the final push into overt illness. The condition was one of reaction to heart disease and incapacity, in a woman whose whole life had been one of weary toil in other people's hotels and boarding-houses. Electroconvulsive therapy was deemed inadvisable after the cardiologist's report had been received, although she had had four shocks previously with no bad results. Rest, sedation, massage and encouraging talks remained as therapeutic measures. The outlook for the patient was only fair, unless she lived at peace with her heart disease and obtained the pension. She said that she did not want to die, but could not stand the strain of living at present.

Obsessive Illnesses.

The first of a group of patients suffering from obsessive illnesses was a man, aged forty-four years, a labourer, who had been admitted to the clinic because of obsessional and worrying thoughts that ran through his mind. The present attack had started three weeks before his admission to the clinic, after a rather heavy bout of drinking. He saw himself cutting his throat and shooting himself, and felt that he could harm others also. He feared the thoughts, and was afraid that they would drive him mad. As a result he was depressed and introspective. His wife was one of the persons whom he could see himself killing. He had lost his interest and could not work. He tried to free his mind of the ideas by reading and by taking medicine which the chemist gave him and which had been successful on former occasions. Now those practices had failed to give him ease, and for the third time he had come to the clinic with a similar complaint. He had a phobia attaching itself to guns and sharp instruments. His mother had been a diabetic who died of gangrene, his sister was "nervy", and a brother and a sister were in a mental hospital. He had been a coalminer and a prawncatcher, and was addicted to alcohol. He did not get on well with his wife, who objected to his obsessive ideas and to his drinking habits. She was afraid that he would harm her and the children, and she refused him intercourse. He said that his trouble might have begun when he was aged ten years, for then he had practised mutual masturbation with a girl, for which he was caught and punished. He was jealous of his wife and had taken legal action against another man. It was pointed out that there might have been some repression of guilt and fear when he was caught and punished at the age of ten years, and that some of that repressed affect was causing the obsessive thought to arise in his conscious mind. Alcohol appeared to be a precipitating factor in the onset of his acute attacks, and he stated that his marital troubles (his wife was unfaithful) made him need the alcohol more. Electroconvulsive therapy had helped him

on previous occasions, but at present he feared the treatment, and insight therapy was being tried. Permanent cure was not thought likely, but at times his symptoms did lift satisfactorily.

The second patient of the group was a single man, a labourer, aged twenty-one years, who had been admitted to the clinic on July 25, 1950, because he was disturbed in the presence of fires, and had a compulsive desire to burn his hair off. For five months he had been worried by the presence of fires, and had a fear that he might be burnt. He was unable to boil water lest his hair catch fire. He was introspective and preoccupied with his personal feelings, spoke in a foolish manner, objecting to people clearing their throats, was depressed, and derived satisfaction in tearing his clothes and in striking persons who offended him. Compulsively he would place his hair in acid if acid was about; that would cause baldness. The fears had reached obsessive value, and so affected him that they inhibited all his activities and excluded all else. He had rather a childish outlook on things generally. He lacked judgement, had ideas of reference, and felt that he might become insane if treatment failed to relieve him. His intelligence quotient was 90. Rorschach testings showed signs of an early psychotic condition; schizophrenia was indicated also in this test and in the Wechaler-Bellevue examination. His father was an alcoholic hawker, though formerly a dentist. His mother was a depressive sufferer, and his father's sister had hand-washing mania. His education had taken him to first year at high school at the age of fifteen years, but he frequently was a truant, when he was severely punished by his parents; they used to scream at him and make him afraid. He played football and billiards, and could dance. He was addicted to self-abuse, and had had many irregular liaisons with girls; he had had nine female friendships. The illness had flared up in January, 1950, with the hair-burning temptation; when his hair grew at all, he looked in the mirror and wanted to burn it off, and to throw flour, acid and cement on it. He became very excited and had to do things at once. He was gambling heavily, for him, and put all his money on one horse. He drank strangely, taking ten gins as one in his beer. He had strong urges to poke his eyes out with sharp objects, such as needles, or to rip off his buttons and tear his pockets open. He had wished to put his fingers under a heavy press, and he had given himself electric shocks. It was commented that he was a strange youth, whose personality was very poorly integrated. The early life travelling about the country with a violent and alcoholic father, and his fears of accidents on the road, had not helped him to shape any course in life. He showed aggression in many forms, and often desired to turn it against himself. His phobias had assumed control of his other mental activities, and he had drifted into a condition of lack of interest and apathy, which must place him in the schizophrenic group; he was drifting rapidly into a more severe psychosis. He had had a course of full insulin coma, but there had not been much change in his condition up to the present time (treatment was unfinished). He had made a good social recovery and was usefully employed.

A male student, aged twenty-two years, was then presented. He was an illegitimate child; the father had deserted the single mother, who went to another State to have the child. The child was adopted at the age of one day and taken to Broken Hill by the foster-parent. All care was lavished on him. He was a healthy lad and a good scholar in primary school; but in technical high school he got in with bad company which influenced him not to work. At the age of fifteen years, when at school, he got into the bedroom of a Greek girl every night and slept with her until dawn; then he ran home again. Finally he was chased all over the town by the Greek girl's father, with a large knife, and escaped, but was exhausted and terrorized. He was then brought to Sydney, but refused to study and went with girls of bad reputation. From then onwards the foster-parents did no good with him. He joined the Royal Australian Air Force as wireless air gunner and went to Canada to complete his course. He crashed in the snow there, and was burnt on the hands, which became septic. He returned to Australia, having experienced no war action. He decided to undergo rehabilitation by studying to be a dentist, and he began training in Adelaide to matriculate. He could not concentrate, his memory became bad, and he was restless. He went home to Silverton, where he worked on a poultry farm; his symptoms were tinged by his work. Seven months before his admission to the clinic he developed an obsession that he had dirt and germs on him, and he began to wash his hands, body and legs; the hand-washing mania was fully developed. He was unable to concentrate, to think or to work because of the overwhelming fear. He was apathetic and dull, and there was a chance of suicide. He used a roll of toilet paper at a time and felt that he was dirty; he wet himself, so slow was he at toilet operations. At the present time he was now seeking a good girl by whom he might have a large family. He had a violent temper. The main factors of his history related to his illness were poor heredity, septic hands in Canada and the dirt on a farm. He had a good affective tone. The diagnosis was compulsion neurosis. More recently he had become much concerned by fluff on blankets and on chairs and by dust on plates; he also had some washing mania still. He disliked untidy shirting and tucked his shirt into his trousers smoothly. He felt that he was improving, and he was trying, under suggestion, to live at peace with his worries. The comment was made that an obsession was a persistent irrational thought which the patient knew to be irrational, but from which he could not free his mind. The persistent thought often led to an act, a compulsive act, which the person might realize was foolish, but it had to be done before he could relax the tension of the mind. In an obsession, the individual combated anxiety by means of a complicated ceremonial. He went through a sort of ritual, pronounced magic words, made symbolic gestures; in short, he lent himself to complex activity in order to banish anxiety. The patient shown had been improved a good deal by a course of carbon dioxide and oxygen therapy.

The last patient of the obsessional group was a single woman, aged forty-four years, who had been admitted to the clinic because she was obsessed by fears of dirt and of spreading germs, and by a constantly recurring idea that the universe might be destroyed because of contamination emanating from her. For years active homosexual practices had kept her in a state of periodic ecstasy, but she had given them up four and a half years before coming to the clinic and then had developed strong obsessive ideas, which had gained in force and distribution. She began to wash her hands ceaselessly, feeling that mucus on them, resulting from masturbation, had a death-dealing influence. She later declared that the touch of her hand or body would give people an illness spread by germs, and she believed that she was a disseminator of diseases of unknown origin. The touch of her unnaturally used hands, in her opinion, had caused the death of thousands of persons. She was also obsessed by the idea that her words might convey the dread contaminations, and that if she told a nurse that the work made her tired, then that nurse would die shortly afterwards. She feared entering a church on a rainy day, because drops of water from her umbrella might fall on the floor and cause it to rot, and that would be a deadly sin. The obsessive ideas had reached the stage where she felt that she might have contaminated the insects, birds *et cetera*, and her evil touch was such that the sun and the whole universe would pass into oblivion. The ideas had really gained strong delusional status, and it was possible that she would have to be certified ultimately. So far she had preserved some social graces, behaved well and worked usefully, but she had asked to be sent to a mental hospital for the protection of others. She hoped after that to be able to go out and earn her living usefully. Her family history was bad. Her father had been cruel to her, beaten her, put her out to service and taken away all her wages, and ultimately he had drunk himself to death. Her mother had died of consumption in the patient's earlier years, and a stepmother had been harsh towards her. Her sister had died of pulmonary tuberculosis. She ran away from home eventually and was lady's companion to one woman for eighteen years; then the woman went into a mental home. The patient had practised masturbation, including mutual masturbation with other girls, since the age of seven years. Later she used to stand and urinate in a male fashion. When a lady's maid she engaged in homosexual practices with her employer and would fall into a state of ecstasy. She acted in that fiendish manner for eighteen years and then her employer passed into insanity. The patient was entirely homosexual; men had no appeal for her, but at times she had permitted intercrural intercourse. During her own acts of masturbation she called on phantasy and imagined herself in a male role. She had more recently acted as housekeeper to a single man, a clergyman, who had to dispense with her services, and finally she had looked after a prostatically stimulated octogenarian, who had since died. It was pointed out that the patient had a strong history of psychopathy and her father's harshness had turned her away from male interests. She had been a strong homosexual type, who preferred tall well-built women. The repression of the guilty affect from her sexual play as a child was now freed and had attached itself to the strongly obsessive ideas. She probably could not be cured, but she should be kept in as good an environment as possible and for as long as her social behaviour permitted.

Her fears were not for herself, but were all expressed in terms of her desire never to be guilty of harming any other person. She said that she believed that one day the medical profession would discover the diseases which she had and which were capable of such fatal and far-flung disasters, attaining really to atomic proportions.

Neurasthenia.

A single man, aged fifty-eight years, was presented suffering from neurasthenia. He had been admitted to the clinic complaining of easily induced fatigue with vague aches and pains in the eyes. For a long time he had shut himself up in darkened rooms and he wore a strange dark-glass apparatus at all times. That had been reduced by the clinic to one set of dark glasses. So often did he complain of his eyes and forehead that eye and ear, nose and throat specialists had examined him on more than one occasion, with negative results. He complained of an unendurable fatigue, which he claimed was set up by the extreme sensitivity of his eyes to light; he complained loudly of the trouble and resented doing any work at all. Sedation, he claimed, gave him but little sleep, yet he obtained six or seven hours' sleep nightly. He was hypochondriacal in other matters. Investigation of his background showed that his father had died of ulcerated stomach and that his mother had succumbed to childbirth. He had had a reasonable childhood and was a normal scholar, but at the age of fourteen years he began to take epileptoid turns and was later discharged from the Railways Department on that count. He was very attached to his mother, and on her death he transferred the fixation to a single sister, with whom he lived. He had a small income from a legacy and had done some casual boot repairing. Heavy masturbation had been his sex outlet and he had a good deal of remorse over it; that was the Freudian basis for his trouble. He had an inadequate personality; the dismissal from the Railways Department for fit-taking and the owning of a small income had been powerful factors justifying him in leading his present idle life. He was on that account unlikely to be cured. He was immature in outlook, being so religious that he regarded sex and all pertaining thereto as filth.

Facial Hemiatrophy: The Parry-Romberg Syndrome.

A married woman patient was shown who had been admitted to the clinic on June 13, 1938, suffering from a depressive state. She had "Cardiazol" convulsion treatment and was discharged from the clinic very much improved on July 24, 1948. On August 16, 1943, she reported and complained of "inward worrying" and severe headaches. Facial asymmetry was noticed with wasting in the right submalar region. An X-ray examination showed mucosal thickening on the maxillary antra, with slight haziness of the frontal sinuses. On August 30, 1943, fibrillation was noticed in the right cheek and the tongue was protruded to the right. About one month later fibrillation was noticed in the neck on the same side. She was examined again on November 20, 1950, and the condition was much the same as in 1943. The comment was made that the syndrome was characterized by a progressive atrophy of all the tissues, including bone, and was said to undergo spontaneous arrest after a few years. There was no sensory loss or paralysis. The condition was usually confined to one or more branches of the trigeminal nerve, but had been observed to extend to the upper cervical nerves. No pathological condition had been discovered to account for the atrophy. It was more common in females. The only treatment was injection of paraffin to fill the submalar hollow.

Public Health.

THE SOUTH AUSTRALIAN TUBERCULOSIS ASSOCIATION.

THE seventh annual report of the South Australian Tuberculosis Association, Incorporated, for the period ended June 30, 1950, is an encouraging document. The report covers a period of eighteen months instead of twelve months as it did before. In 1949 the constitution of the association was amended, and provided *inter alia* that the financial year should end on June 30 instead of December 31.

The aspect of the report to which attention is specially directed has to do with what is known as Bedford Industries, an organization designed to secure the gradual rehabilitation of sufferers from pulmonary tuberculosis so that they may once more take their places in industry. It should perhaps be explained that the South Australian Tuberculosis Association is controlled by a governing committee of which Sir Henry Newland is President. The Medical Director and Chairman of the Executive is Dr. D. R. W. Cowan. The Board of Management of Bedford Industries comprises Mr. L. M. Anderson, Chairman, Sir C. Stanton Hicks, Dr. D. R. W. Cowan, Dr. A. C. Savage, Mr. H. R. Gledhill, Mr. R. K. Wood and Mr. T. H. Brown. There are two government representatives on the Board of Management, Dr. J. W. Rollison, the Director-General of Medical Services, and Mr. G. E. Scantlebury, the Secretary of the Hospitals Department.

It was in 1939 that Dr. D. R. W. Cowan made the first efforts to establish a rehabilitation centre, where men able to undertake light work could begin to fit themselves for reentry into productive fields after long breaks due to illness. Unforseen difficulties were encountered and plans could not be brought to fruition until April, 1945, when the South Australian Tuberculosis Association took over from Mr. A. V. Pimlott, a small woodworking establishment conducted by him in rented premises at East Glenelg. The business was registered under the name of Bedford Industries and this marked the beginning of the Tuberculosis Association's industrial venture. Mr. Pimlott's services were retained as manager, and several men, both ex-servicemen and civilians, were engaged for varying periods of time. Orders were readily obtained, and since the start have greatly exceeded the capacity of the works. Over the first fifteen months, the number of articles produced exceeded 23,000. The number of permanent and part-time employees grew by degrees until in 1949 there was an average of over 20. In the premises at Glenelg there were many disadvantages. The factory area was small and the layout was not altogether suitable. The plant was not sufficiently extensive and experience had to be gained by newcomers to the industry. As a result losses were inevitable, and from 1945 to 1949 these were borne by the association. The State Government was impressed by the work that was being done and undertook to subsidize the erection of a modern factory to the extent of £20,000 on a pound for pound basis. It also undertook to provide an annual grant in the form of a subsidy on possible losses when the complete scheme went into operation. Governments are so often blamed for not having done their duty in public health matters, that emphasis should be laid on the clear-sighted action of the South Australian Government at that time.

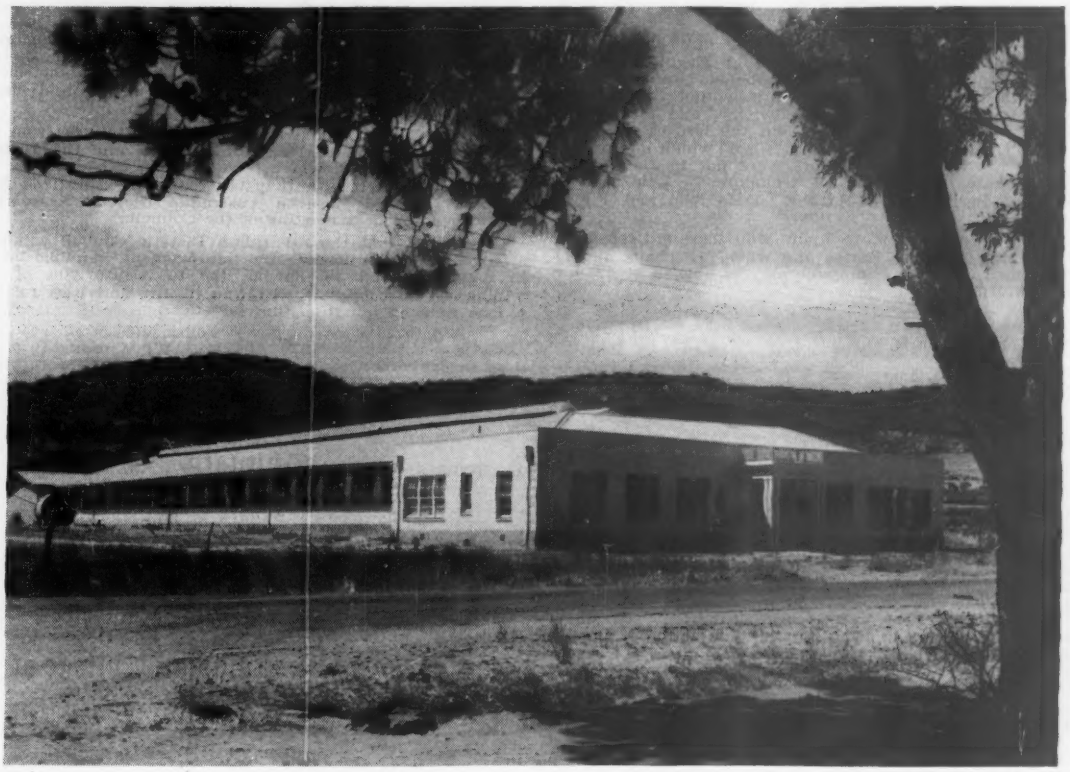
At the end of 1946 a strong committee under the chairmanship of the late Mr. F. W. Cornell launched a public appeal for funds. With the money subscribed by South Australian firms and by individual members of the community and with the appropriate government subsidy a magnificent new factory has been built. This factory stands at an elevation of 165 feet about four miles from the city of Adelaide and six miles from the sea. It stands on four and a half acres of land held by the Crown. The land has been dedicated for public health purposes and the control and management have been vested in the association. Recently the association has acquired a further area of four and a half acres immediately adjoining the factory. This land is being held for future development and it is hoped later to erect a number of homes for the accommodation of personnel. The present plans provide for the erection of annexes for fresh lines of manufacture, and particularly for the employment of female ex-patients if a sufficient number are available to justify the outlay in the matter of plants. The new factory is situated in a non-industrial area and has been designed to harmonize with the architectural features of nearby public buildings. It should be noted that in recognition of his efforts as founder of the industry Dr. D. R. W. Cowan has been honoured by the naming of the block as the Cowan Building.

The Cowan Building has been planned so as to give ideal working conditions. In the factory block alone there are some 10,000 square feet of space and amenity rooms, and administrative offices are designed in accordance with modern practice. The work undertaken by male employees is in the manufacture of wooden articles, and for this purpose modern machinery has been installed. Special measures are undertaken to cope with the presence of dust. At the present time some female employees are engaged in the manufacture of such household requirements as pillow cases. The goods manufactured by male employees at present include bath mats in many sizes, wash boards, children's play-pens, clothes airers and step-ladders. Pinus from the South Australian government forests is used in large quantities, and selected hard-woods are handled for the manufacture of a number of lines. All articles are sold at current market prices and all labour is engaged at

current award rates. This means that the factory is being conducted on a purely competitive basis.

When a sufferer from tuberculosis is considered to be well enough to engage in some occupation he is examined carefully before he is allowed to start work at Bedford Industries. The length of his work periods is defined and suitable periods of rest are enjoined. A careful check is kept on his medical condition. The persons employed are ex-patients from the metropolitan area and approved patients from various hospital sanitoria. Everyone employed in and around the factory has been a sufferer from tuberculosis.

The plans for work at Cowan Building include the introduction of new lines from time to time. It is pointed out in the report that such developments must come step by step and only as demand exists for the product.



The South Australian Tuberculosis Association expresses the opinion that with four divisions of the Australian Tuberculosis Association now functioning in four States of the Commonwealth, the lead given in Adelaide will be followed in other States. A start has already been made in Western Australia where a lease of an established business has been secured and plans to erect premises at an early date have been made.

From the foregoing it will be apparent to readers that most important work is being carried on at Adelaide. Medical practitioners who are visiting that city would be well advised to see for themselves what is being done at Bedford Industries.

The Secretary of the South Australian Tuberculosis Association is Mr. H. C. Dridan and its offices are at Gawler Chambers, North Terrace, Adelaide.

Correspondence.

THE GROCERS' COMPANY SCHOLARSHIP.

SIR: It is desired to advise you that information has been received that the Grocers' Company, Grocers' Hall, London, E.C.2, will offer one scholarship each year, tenable for two years, of the value of £450 for the first year and £500 for the second year, subject to a satisfactory report from the assessors appointed by the Court of the Grocers' Company, with the object of encouraging original medical research. An additional sum not exceeding £50 a year for each scholar will be provided to defray the cost of special apparatus, of materials, and of laboratory and other general expenses.

Under the terms of tenure the scholarship shall begin on the first day of September, and no remunerative employment is to be undertaken without the express sanction of the Court. The applicants must be British subjects and under thirty-five years of age.

Each applicant must supply the following information: (i) The nature of research which he proposes to undertake. (ii) Reasons for considering the suggested research of importance and capable of completion within the period of the tenure of the scholarship. (iii) The institutions, laboratories and place or places in the United Kingdom wherein or whereat he proposes to carry out the work. (iv) Whether approval of the head of the laboratory in which the work will be carried out has been obtained.

Would you be good enough to bring this information to the notice of your readers? Full details are obtainable from the Grocers' Company, Grocers Hall, Princes Street, London, E.C.2.

Yours, etc.,
A. J. METCALFE,
Director-General of Health.

Canberra,
January 23, 1951.

THE COMMONWEALTH JUBILEE NUMBER.

SIR: Sir Thomas Hurley is to be complimented upon his spirited and comprehensive review of this half-centry of Australian surgery. May I suggest, however, that the phrasing of his comments upon plastic surgery might lend credence to the interpretation that Sir Harold Gillies took no part in the training of Australian plastic surgeons during World War II? As I can cordially testify, the Sir Harold Gillies of Rooksdown House, Park Prewett (from 1940 until

today), is as great and stimulating a teacher as was the Gillies of the Sidcup era. I feel sure that Sir Thomas would wish to be among the first to acknowledge the great debt which Australian surgery owes to this most gifted doyen of plastic surgeons.

Yours, etc.,

185 Macquarie Street, Sydney,
January 9, 1951.

KENNETH W. STARR.

SIR: I was interested in the article on obstetrics and gynæcology on page 63 of the Jubilee number of our journal. It mentions that at the turn of the century the practice of obstetrics may be said to have been on a high plane of efficiency.

It may not be generally known that my late father, Dr Alexander Buttner, who practised at East Melbourne, Victoria, from the late seventies of the last century until his death in 1914, was one of the first, if not the first, in Victoria to apply antisepsis in midwifery, and during his practice confined 5000 women without the loss of one. He first used "Lysol" and subsequently "Alsol" which he imported himself, as these medicaments were not in those days procurable here. He learnt his technique in Leipzic, was an M.D. of Berlin and M.R.C.P.E., F.R.C.S.E. and F.F.P. and S.G.

It would be of interest to know who used antisepsis in obstetrics first in other States and when. Perhaps among the older men there are some who may be able to give some reliable information.

Yours, etc.,

131 Domain Road, South Yarra, S.E.1, Victoria,
January 15, 1951.

F. A. BUTTNER.

TREATMENT OF ECZEMA.

SIR: Under the heading "Treatment of Eczema" you published a letter from Dr. Britten (MEDICAL JOURNAL OF AUSTRALIA, January 20, 1951) advocating certain arsenical mixtures. May I sound a warning in addition to the one he quotes? There is grave danger in continued administration of arsenic. The worst case of chronic arsenical poisoning I have seen was an infant admitted to the Children's Hospital some years ago having taken a mixture similar to the one he quotes and presumably from the same source. Only after much strenuous treatment was this baby's life saved. Infantile eczema is due to many obscure dietetic and absorptive factors and inherited tendencies, and cannot be cured by a bottle of medicine, but by good nursing and observation accompanied by all the wiles and wisdom and experience of a sound physician it can be alleviated and may be cured.

The use of prescriptions such as those advocated without constant supervision can only be followed by tragedy.

Yours, etc.,

12 Collins Street, Melbourne, C.1,
January 22, 1951.

ARTHUR J. DAY.

A DISCLAIMER.

SIR: I have been embarrassed lately in that a Press report was published both in Sydney and Melbourne wherein my name was mentioned in connection with "Cortone" (cortisone) treatment. The latter was done without my knowledge or permission.

Since then, the newspaper concerned apologized to the Medical Secretary of the Victorian Branch of the British Medical Association. I shall be grateful if you will publish this note.

Yours, etc.,

34 Queen's Road, Melbourne,
January 21, 1951.

CLARENCE M. MARSHALL.

THE AUSTRALIAN DIETETIC COUNCIL.

SIR: On May 19, 1950, the inaugural meeting of the Australian Dietetic Council was held at the Royal Melbourne Hospital and the following office bearers were elected: *President*, Miss Joan Woodhill, B.Sc.Agr., M.S., A.I.D., Research Fellow, King George V and Queen Mary Maternal and Infant Welfare Foundation, Sydney. *Vice President*, Mrs. Audrey Cahn, B.Sc.Agr., Lecturer in Nutrition, University of Melbourne. *Secretary-Treasurer*, Miss Winifred Wilson, B.A., A.I.D., Dietitian, New South Wales Department of Public Health.

The objects for which the Council is established include the following: (*a*) to foster and develop dietetic work in Australia; (*b*) to further the knowledge of and promote efficiency in dietary work; (*c*) to approve standards for the educational and scientific qualifications of students in dietetics; (*d*) to facilitate the communication and the interchange of ideas with organizations dealing with dietary work in Australia and overseas and to bring about closer cooperation among dietitians; (*e*) to advise and assist associated bodies in any matter of common interest.

At present the Council includes representatives from the Dietetic Association of New South Wales and the Dietetic Association, Victoria, which have been in existence for the past ten years. It is anticipated that representatives from the newly-formed Western Australian Dietetic Association will attend the next meeting of the Council.

It is hoped that the Australian Dietetic Council will play a part in the maintenance and improvement of public health in Australia, and we should like to assure you of our willingness to cooperate with you in any activities relating to the field of nutrition and dietetics.

Yours, etc.,

52 Bridge Street, Sydney.
January 3, 1951.

W. WILSON,
Honorary Secretary.

Obituary.

THOMAS PATRICK BYRNE.

WE regret to announce the death of Dr. Thomas Patrick Byrne, which occurred on January 26, 1951, at Coogee, New South Wales.

The Royal Australasian College of Physicians.

CORTISONE.

THE Royal Australasian College of Physicians has been approached by a firm of Australian importers of drugs from abroad and has been asked to act in an advisory capacity in the allocation of such supplies of cortisone as may become available in Australia through this firm.

Preferences in allocation will be given to those hospitals or institutions which are carrying out investigational work under the supervision of committees specially appointed for this purpose. Supplies of the drug are limited, and allocation will only be made to a private practitioner for a particular case, details of which are submitted to and approved by the College. Applications for the drug should be submitted in writing to the Honorary Secretary, The Royal Australasian College of Physicians, 145 Macquarie Street, Sydney, and should include the following details: (i) Essential clinical data of each case or the projected research. (ii) An estimate of the amount of the drug required in the first instance. (iii) An undertaking to supply a full report of results of treatment in each particular case or research project.

It is anticipated that in this way sufficient clinical information will become available to establish the most beneficial use of the drug in this country.

It is understood that some supplies of cortisone are available in Australia from sources other than the firm which has approached the College in this matter. For these the College can accept no responsibility.

Naval, Military and Air Force.

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 78, of December 14, 1950, and Number 4, of January 11, 1951.

NAVAL FORCES OF THE COMMONWEALTH.

Permanent Naval Forces of the Commonwealth (Sea-Going Forces).

Appointment.—Peter Winton Kentwell is appointed Surgeon Lieutenant (D) (for short service), dated 16th October, 1950.

Loan to Royal Navy for Service and Training.—The following to be loaned to the Royal Navy for service and training: Surgeon Lieutenant-Commander Kenneth Charles Armstrong, dated 17th June, 1950.

Citizen Naval Forces of the Commonwealth.

Royal Australian Naval Reserve.

Appointments.—Austen Stewart Ferguson (Surgeon Lieutenant-Commander, Royal Australian Naval Volunteer Reserve) is appointed Surgeon Lieutenant-Commander, with seniority in rank of 2nd August, 1949, dated 12th October, 1950. Desmond Albert Cooper is appointed Surgeon Lieutenant (on probation), dated 9th October, 1950.

AUSTRALIAN MILITARY FORCES.

Royal Australian Army Medical Corps.

VX700127 Honorary Captain E. S. R. Hughes is appointed from the Reserve of Officers and to be Captain and Temporary Lieutenant-Colonel, 26th October, 1950. VX700128 Honorary Captain R. G. Webb is appointed from the Reserve of Officers, and to be Captain and Temporary Major, 26th October, 1950.

Citizen Military Forces.

Northern Command: First Military District.

Royal Australian Army Medical Corps (Medical).—1/39091 Honorary Captain D. J. Chapman is appointed from the Reserve of Officers, and to be Captain (provisionally), 31st August, 1950. 1/39096 Honorary Captain E. F. McGuinness is appointed from the Reserve of Officers, and to be Captain (provisionally), 16th October, 1950. The provisional appointment of 1/39085 Captain G. C. T. Kenny is terminated, 29th September, 1950.

Eastern Command: Second Military District.

Royal Australian Army Medical Corps (Medical).—2/146572 Captain G. P. Dynon is appointed from the Reserve of Officers, 6th September, 1950. To be Captains (provisionally), 5th October, 1950: 2/127019 Roy Herbert Donald Bean, 2/127807 John Brock Westphalen and 2/127808 Brian Luden Vardon McLaughlin.

Southern Command: Third Military District.

Royal Australian Army Medical Corps (Medical).—3/101010 Captain (provisionally) S. H. Watson is seconded whilst undergoing post-graduate studies in the United Kingdom, 23rd June, 1950. 3/50090 Lieutenant-Colonel A. J. M. Sinclair is appointed from the Reserve of Officers and to be Temporary Colonel, 23rd August, 1950. 3/101805 Honorary Captain J. P. Morris is appointed from the Reserve of Officers and to be Captain (provisionally), 18th September, 1950.

To be Major (provisionally), 20th October, 1950.—3/101005 Captain T. D. Hollywood.

Central Command: Fourth Military District.

Royal Australian Army Medical Corps (Medical).—4/31956 Captain L. C. Lum is seconded whilst undergoing post-graduate studies in the United Kingdom, 23rd October, 1950.

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED JANUARY 13, 1950.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.[2]	Western Australia.	Tasmania.	Northern Territory.[2]	Australian Capital Territory.	Australia.[2]
Ankylostomiasis	*	..	..	..	..	..	..	..	..
Anthrax	*	..	..	..	..	..	..	..	..
Beriberi	*	*	*	*	*	*	..	*	..
Bilharziasis	*	..	..	..	..	..	..	..	..
Cerebro-spinal Meningitis	2(1)	2(2)	..	..	1	..	..	..	5
Cholera	..	..	..	..	..	..	..	..	..
Coastal Fever(*a*)	*	*	*	*	*	*	*	*	..
Dengue	*	*	*	*	*	*	..	*	..
Diarrhœa (Infantile)	..	..	5(5)	..	..	..	..	..	5
Diphtheria	8(6)	1	7(4)	1	3(3)	..	..	..	20
Dysentery (Amœbic)	*	1(1)	..	..	..	..	..	..	1
Dysentery (Bacillary)	*	..	2(2)	..	..	..	*	..	2
Encephalitis Lethargica	1	..	..	..	..	..	..	..	1
Erysipelas	*	*	*	1(1)	*	*	..	..	1
Filariasis	*	..	..	..	..	..	..	..	..
Helminthiasis	*	..	*	*	*	*	*	*	..
Hydatid	*	..	*	*	..	*	*	..	..
Influenza	*	*	*	..	*	*	..	*	..
Lead Poisoning	*	*	..	*	*	*	*	*	..
Leprosy	..	1(1)	..	..	..	..	..	..	1
Malaria(*b*)	*	..	1(1)	..	..	..	..	..	1
Measles	*	*	*	213(194)	*	*	..	..	213
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	60(27)	6(1)	24(6)	19(15)	..	18(6)	..	..	127
Psittacosis	*	..	*	..	*	*	*	*	..
Puerperal Fever	1	..	..	..	..	..	..	..	1
Rubella(*c*)	*	*	..	*	1(1)	*	..	..	1
Scarlet Fever	13(2)	16(10)	9(3)	4(4)	3(1)	1(1)	..	..	46
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	*	..	..	*	*	*	*	*	..
Trachoma	*	..	*	*	*	*	*	*	..
Tuberculosis(*d*)	63(51)	27(26)	12(8)	7(4)	3(2)	5(2)	..	..	117
Typhoid Fever(*e*)	..	..	1(1)	..	..	..	..	..	1
Typhus (Endemic)(*f*)	..	..	..	..	..	..	..	..	..
Undulant Fever	*	2	..	*	*	*	*	*	2
Weil's Disease(*g*)	*	*	2	*	*	*	*	*	2
Whooping Cough	*	*	*	4(1)	*	*	..	..	4
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] The form of this table is taken from the *Official Year Book of the Commonwealth of Australia*, Number 37, 1946–1947. Figures in parentheses are those for the metropolitan area.
[2] Figures not available.
[3] Figures incomplete owing to absence of returns from the Northern Territory.
* Not notifiable.
(*a*) Includes Mossman and Sarina fevers. (*b*) Mainly relapses among servicemen infected overseas. (*c*) Notifiable disease in Queensland in females aged over fourteen years. (*d*) Includes all forms. (*e*) Includes enteric fever, paratyphoid fevers and other Salmonella infections. (*f*) Includes scrub, murine and tick typhus. (*g*) Includes leptospiroses, Weil's and para-Weil's disease.

Western Command: Fifth Military District.

Royal Australian Army Medical Corps (Medical).—5/32061 Major (Honorary Lieutenant-Colonel) J. M. M. Drew, O.B.E., is appointed from the Reserve of Officers and borne supernumerary to the authorized establishment of Majors with pay and allowances of Captain (at his own request), 2nd August, 1950.

Reserve Citizen Military Forces.

Royal Australian Army Medical Corps.

1st Military District: To be Honorary Captains.—Geoffrey Charles Treadgold Kenny, 30th September, 1950, and Edward Harry Brown, Ian Keith Birchley and Bruce Geaney, 27th October, 1950.

4th Military District: To be Honorary Captain, 27th October, 1950.—Franklyn Gilbert Bell.

5th Military District: To be Major and Honorary Lieutenant-Colonel, 1st August, 1950.—John Murray Macfarlane Drew, O.B.E.—(Ex. Min. No. 6—Approved 4th January, 1951.)

ROYAL AUSTRALIAN AIR FORCE.

Citizen Air Force: Air Force Reserve.

Medical Branch.

The following former officers are appointed to commissions with the rank of Flight Lieutenant: D. Gordon (276870), 23rd August, 1950, G. F. Salter (253256)), 25th September, 1950.

The following are appointed to commissions with the ranks indicated: (Flight Lieutenant) (Temporary Squadron Leader) John James Woodward (257792), 21st September, 1950 (Flight Lieutenant) Evan Ross Landeg Williams (277570), 4th August, 1950, (Pilot Officer) Gordon Murrie (257795), 28th July, 1950.

The following are appointed to commissions with the rank of Flight Lieutenant: John Andrew Edye (266715), 8th August, 1950, Brian Eric Frecker (267894), 16th August, 1950, Rhys Ivor Johns (267898), 30th October, 1950.

Flight Lieutenant C. Roe (277273) is promoted to the temporary rank of Squadron Leader, 26th October, 1950.

The appointment of Temporary Squadron Leader S. F. McR. Yeates (272046) is terminated, 30th October, 1950.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Anderson, Gerard James, M.B., B.S., 1951 (Univ. Sydney), Lewisham Hospital, Lewisham.

Harrell, Francis Charles, M.B., B.S., 1951 (Univ. Sydney), Royal North Shore Hospital, St. Leonards.

Soper, Henry Lewry, M.B., B.S., 1951 (Univ. Sydney), 9 Tedman Parade, Sylvania.

Worling, Babette Josephine, M.B., B.S., 1951 (Univ. Sydney), 2 Eric Road, Artarmon.

Enis, Zenon, registered in accordance with the provisions of section 17 (1) (c) of the *Medical Practitioners Act,* 1938-1950, 63 Queen Street, Ashfield.

Johnson, Wilga Miriel, M.B., B.S., 1951 (Univ. Sydney), Lithgow District Hospital, Lithgow.

Owen, Francis Septimus, M.B., 1950 (Univ. Sydney), Royal North Shore Hospital, St. Leonards.

Jones, Bruce Littlewood, M.B., B.S., 1951 (Univ. Sydney), 41 Crordon Road, Epping.

Cross, Douglas Oakeley, M.B., B.S., 1951 (Univ. Sydney), 43 Gordon Street, Balgowlah.

Sharp, Andrew Cruickshanks Fallow, M.B., B.S., 1951 (Univ. Sydney), Inverell District Hospital, Inverell.

Shiels, Eleanor Mary, M.B., B.S., 1951 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

Gibson, David Bruce, M.B., B.S., 1951 (Univ. Sydney), District Hospital, Camden.

Reid, Bevan Leslie, M.B., B.S., 1951 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

Waugh, Ian Francis, M.B., B.S., 1948 (Univ. Sydney), 9 Dalton Road, Mosman.

Bull, Alan Stuart, M.B., B.S., 1950 (Univ. Sydney), 491 New South Head Road, Double Bay.

Hodgkinson, Anthony Hugh Taylor, M.B., B.S., 1951 (Univ. Sydney), 16 Kitchener Street, Maroubra.

Rivett, Ronald Albert, M.B., B.S., 1951 (Univ. Sydney), 148 Edinburgh Road, Castlecrag.

Lewis, Ronald Gordon, M.B., B.S., 1951 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

Medical Appointments.

Dr. R. J. Farnbach has been appointed a member and Chairman of the Patent Medicines Advisory Committee of Victoria, pursuant to the provisions of Section 3 (2) (*a*), (*b*), (*c*) and (*d*), respectively, of the *Health (Patent Medicines) Act,* 1942.

Dr. J. H. Russell has been appointed Director of Tuberculosis, Department of Public Health, Tasmania.

Dr. M. T. Burnell, Dr. J. H. Stace and Dr. A. W. Wall have been appointed honorary anæsthetists at the Royal Adelaide Hospital, Adelaide.

Dr. D. A. Game has been appointed Outpatients' Registrar at the Royal Adelaide Hospital, Adelaide.

Diary for the Month.

FEB. 13.—New South Wales Branch, B.M.A.: Executive and Finance Committee.

FEB. 22.—Victorian Branch, B.M.A.: Executive Meeting.

FEB. 22.—South Australian Branch, B.M.A.: Branch Meeting.

FEB. 23.—Queensland Branch, B.M.A.: Council Meeting.

FEB. 26.—Federal Council, B.M.A. in Australia: Meeting at Melbourne.

FEB. 27.—New South Wales Branch, B.M.A.: Ethics Committee.

FEB. 28.—Victorian Branch, B.M.A.: Council Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney)—All contract practice appointments in New South Wales.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federal Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178 North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia; Medical Officer, South Australian Railways.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Norseman Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

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